5. HUMAN HEALTH BASELINE RISK ASSESSMENT

5.1 Introduction

This Baseline Risk Assessment (BRA) is divided into two evaluations: a human health risk assessment (Section 5) and an ecological risk assessment (ERA) (Section 6). The human health risk assessment approach used in this BRA is based on the Environmental Protection Agency's (EPA's) Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual (Part A) (EPA 1989), the Idaho National Engineering Laboratory (INEL) Track 2 Guidance document (DOE-ID 1994), and the INEL Cumulative Risk Assessment Guidance Protocol (LMITCO 1995). Similarly, the ERA approach used in this assessment is based on the INEL Screening-Level Ecological Risk Assessment (SLERA) guidance document (VanHorn et al. 1995).

Preliminary evaluations of both human health and ecological risks at Waste Area Group (WAG) 9 have been completed as part of the Work Plan for Operable Unit 9-04 Comprehensive RI/FS (Lee et al. 1996). A preliminary evaluation of WAG 9 human health risks and ecological risks were conducted in the WAG 9 Screening and Data Gap Analysis (SDGA) and the WAG 9 SLERA, respectively. The SDGA and the SLERA are found in Section 2 and Section 4 of the Work Plan for Operable Unit 9-04 Comprehensive RI/FS (Lee et al. 1996).

5.1.1 Scope and Objectives

A discussion of general comprehensive risk assessment methodologies is presented in the INEL Cumulative Risk Assessment Guidance Protocol (LMITCO 1995). As discussed in the INEL Cumulative Risk Assessment Guidance Protocol document, the analysis methods used in INEEL comprehensive risk assessments are often different from the analysis methods used in INEL Track 1 and Track 2 risk assessments (DOE-ID 1994). In general, the differences between the two types of analyses are present because comprehensive risk assessments are meant to analyze risks produced by multiple release sites within a WAG, while Track 1 and Track 2 risk assessments are only meant to analyze risks from one release site at a time.

In order to satisfy the broader objective of INEEL comprehensive risk assessments, the INEL Cumulative Risk Assessment Guidance Protocol (LITCO 1995) recommends analyzing risks produced through the air and groundwater exposure pathways in a "cumulative manner." A cumulative analysis of these two exposure pathways involves calculating one WAG-wide risk number for each contaminant of potential concern (COPC) in each air and groundwater exposure route (e.g., inhalation of fugitive dust, ingestion of groundwater, etc.). Analyzing the air and groundwater pathways in a cumulative manner is necessary because a receptor can be exposed to the cumulative concentration of groundwater contamination and also exposed to the cumulative effect of the dusts blowing across all release sites within a WAG. Regarding the soil pathway exposure routes (ingestion of soil, ingestion of homegrown produce), the receptor is exposed to only one release site at a time (produce can only be grown in one site at a time). As a result, the guidance protocol recommends that soil pathway exposure be analyzed on a release-site-specific, or a noncumulative basis in the INEEL comprehensive risk assessments.

The details of the "comprehensive" and "cumulative" aspects of the OU 9-04 BRA are discussed more completely in the following sections. In general, this BRA is "comprehensive" because it evaluates risks from all known and potential release sites within WAG 9, and it is "cumulative" because, where the

contamination to a receptor comes from multiple release sites (e.g., air and groundwater exposure pathways), the risks were calculated based on this cumulative effect of the contamination across these sites.

At WAG 9, one release site being investigated has been classified as a Land Disposal Unit (LDU) under RCRA. This release site is ANL-01A, the Main Cooling Tower Blowdown Ditch (MCTBD). The LDU classification for the MCTBD is based on a pH of 1.86 measured in the discharged effluent in January 1986. The measured pH of 1.86 classified the liquid effluent as being "a corrosive liquid" according to 40 CFR 261.22. A temporary neutralization system was installed in March 1986, and a permanent neutralization tank was installed in October 1986. In October 19, 1995, a letter was submitted to the IDHW-DEQ from DOE requesting that this site be reclassified from a Land Disposal Unit to a Solid Waste Management Unit based on the results of samples collected in 1995. The IDHW-DEQ responded in a letter dated December 18, 1995 and denied the request for removal of the LDU designation for the MCTBD. Ultimately, "The closure of the MCTBD will follow CERCLA under the FFA/CO process with RCRA closure requirements [IDAPA 16.01.05.008 (40 CFR 264 Subpart G)] being strictly applicable." After further review of the FFA/CO agreement by the ANL-W, DOE, EPA, and IDHW WAG 9 managers, the determination has been made that if the MCTBD does not pose a risk greater than those specified in the National Contingency Plan (NCP) the RCRA closure requirements will not be applicable. Thus, the final closure of the MCTBD will depend on the risks calculated for the MCTBD using the CERCLA format.

5.1.2 Human Health Baseline Risk Assessment Tasks

In general, the tasks associated with development of the WAG 9 human health risk assessment is divided up as follows:

- Perform data evaluation, including site and contaminant screening
- Conduct exposure assessment
- Conduct toxicity assessment
- Perform risk characterization
- Utilize risk management decisions
- Conduct uncertainty analysis.

These tasks are described in the following sections.

5.1.2.1 Data Evaluation. All sampling data collected to date at WAG 9 release sites were evaluated to determine whether the data are appropriate and adequate for use in the BRA. This evaluation was conducted in accordance with EPA's Guidance for Data Usability in Risk Assessment (EPA 1992d). Also, as part of this analysis, data sets were plotted, and all were assumed to have lognormal distributions in accordance with EPAs guidance on calculating concentration terms (EPA 1992c).

Data evaluation tasks that were completed as part of the BRA are as follows:

- Identify release sites and co-located facilities that require further evaluation. Co-located facilities are defined as operating or shut down facilities that have the potential for producing future releases of hazardous substances (see Section 5.12.2).

- Review available sampling data for the retained release sites.

- Identify contaminants detected at each release site, and identify the frequency of detection and frequency of exceedance for each contaminant (Section 3.2).

- Identify potential exposure routes (Section 5.3).

- Develop a list of COPCs for each data set, for each site, for use in the risk assessment.

The results of the data evaluation tasks are presented in Section 5.2.

5.1.2.2 Exposure Assessment. The process of exposure assessment quantifies the receptor intake of COPCs for select pathways. The assessment consists of estimating the magnitude, frequency, duration, and route of exposure of chemicals to humans and ecological receptors.

The conceptual site model (CSM) used to develop the BRA exposure assessment is presented in Figure 5-1 and 5-2, and the details of the exposure assessment tasks are presented in Section 5.3.

5.1.2.3 Toxicity Assessment. Toxicity assessment is the process of characterizing the relationship between the dose or intake of a substance and the incidence of an adverse effect in the exposed population. Toxicity assessments evaluate results from studies with laboratory animals or from human epidemiological studies. These evaluations are used to extrapolate from high levels of exposure, where adverse effects are known to occur, to low levels of environmental exposures, where effects can only be predicted based on statistical probabilities. The results of these extrapolations are used to establish quantitative indicators of toxicity.

Health risks from all routes of exposure are characterized by combining the chemical intake information with numerical indicators of toxicity. These health-protective toxicity criteria are obtained through EPA-developed reference doses (RfD) or slope factors (SF). Information used as part of the BRA toxicity assessment is presented in Section 5.9.

5.1.2.4 Risk Characterization. The characterization of risk involves combining the results of the toxicity and exposure assessments to provide a numerical estimate of health risk. This estimate is a comparison of exposure levels with appropriate toxicity criteria, or an estimate of the lifetime cancer risk associated with a particular intake. Risk characterization also considers the nature and weight of evidence supporting the risk estimate, as well as the magnitude of uncertainty surrounding the estimate. The results of the BRA risk characterization process, including risk estimates for each of the retained release sites, are presented in Section 5.10.

5.1.2.5 Risk Management. In risk management, the pathways and risks are evaluated to determine which release sites will be retained for evaluation in the feasibility study. ANL-W, has developed a four step screening process to review the pathways and risks of the retained sites. The risk management review process is presented in Section 5.11.

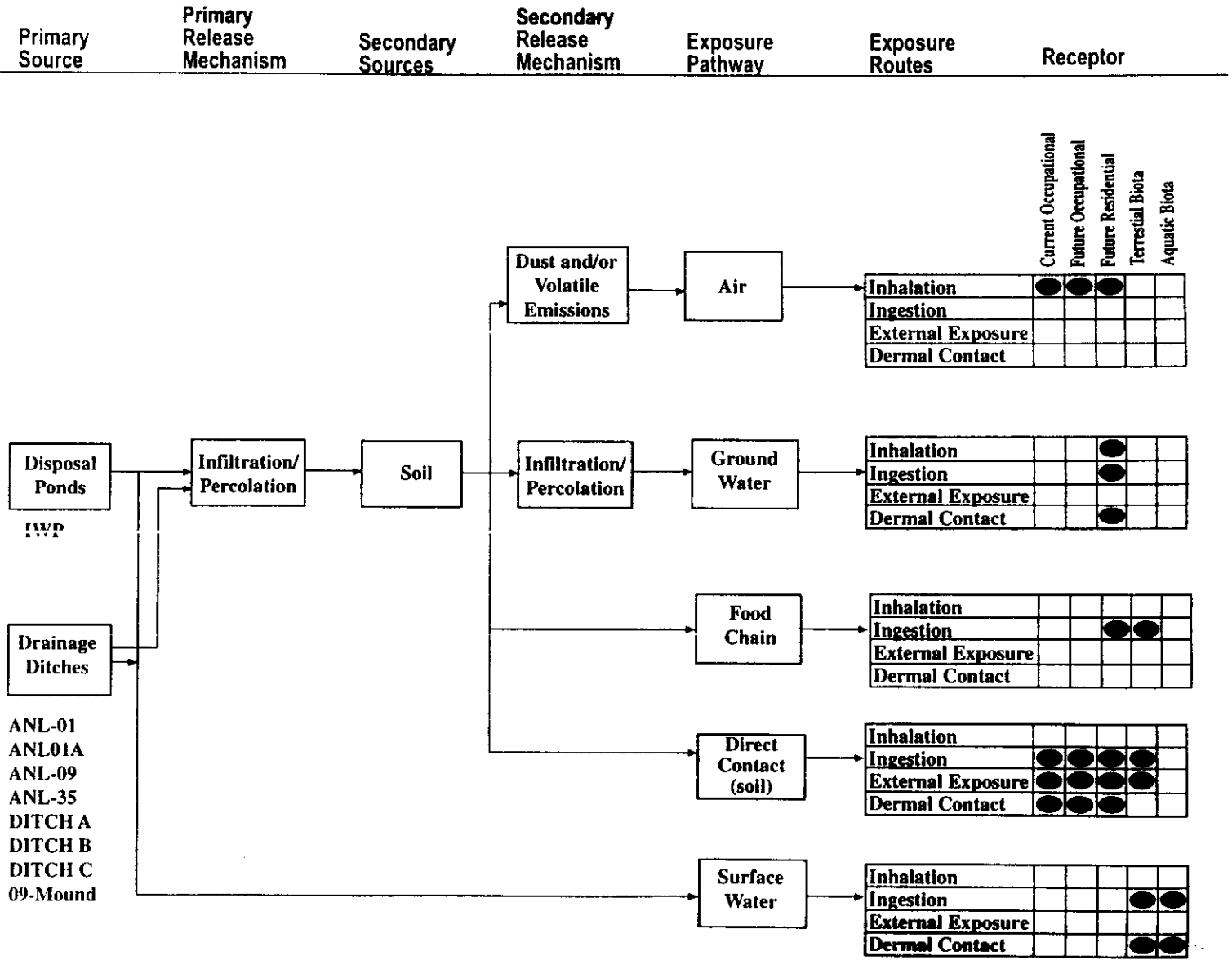

Figure 5-1. Conceptual Site Model of Disposal Ponds and drainage Ditches

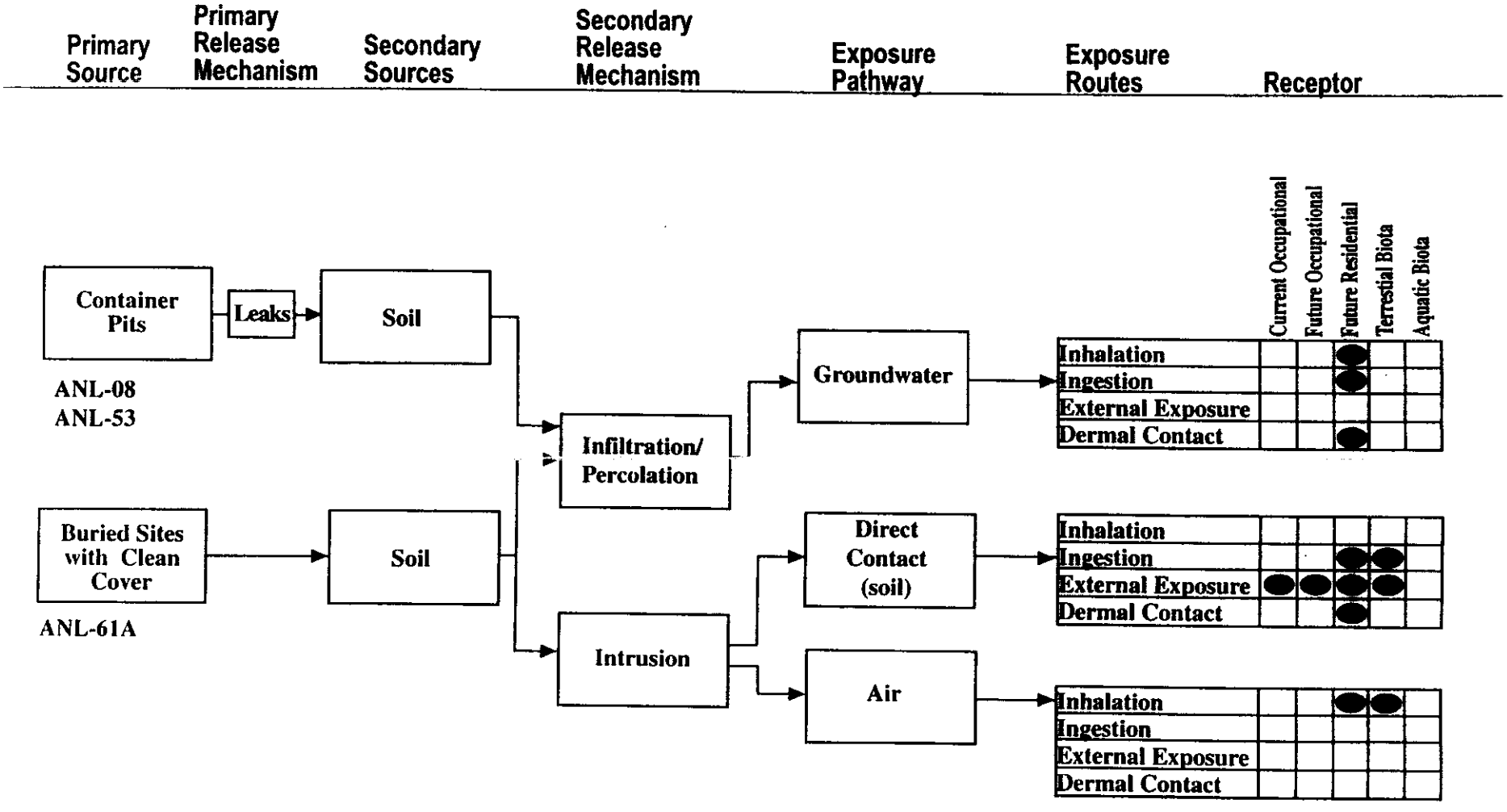

Figure 5-2. Conceptual Site Model of Container Pits and Buried Sites with Clean Cover.

5.2 Data Evaluation

All sampling data collected to date at WAG 9 release sites were evaluated to determine whether the data are appropriate and adequate for use in the BRA. This evaluation was conducted in accordance with EPA's Guidance for Data Useability in Risk Assessment (EPA 1992d). The usable data for each site is then utilized to:

- Screen against backgrounds
- Determine the nature and extent of contamination for each site
- Separate the data in accordance to depth ranges for the various exposure pathways
- Calculate the concentration term.

5.2.1 Data Screening

The steps to complete the contaminant screening are the following:

1. Contaminant concentrations less than or equal to background concentrations were omitted from the risk evaluation. Background concentrations were obtained from Rood et al. (1995). Ninety-five/ninety-five upper tolerance limits for grab samples or composite samples were for soil grab or composite samples, respectively. Contaminants were also eliminated if only one sample exceeded the ninety-five/ninety-five upper tolerance limits, but the concentration was less than the ninety-five/ninety-nine upper tolerance limits for grab samples. If background concentrations were not available for a given contaminant then the contaminant was retained.

2. Six inorganic constituents (i.e., aluminum, calcium, iron, magnesium, potassium, and sodium) are eliminated from analysis in the baseline risk assessment, based on EPA guidance (EPA 1991). For those concentrations of these constituents that exceed background, a footnote will be added explaining why it is believed that these constituents are not from contamination at the site.

Tables 3-3 through 3-18 summarize the maximum concentration of each contaminant found at each site, the background concentrations of contaminants, the frequency of detection, and whether or not the contaminant was eliminated from the risk evaluation. If the contaminant was screened, the justification by step number stated in Section 3.2 is provided in the last column of each table. It should be noted that sludge samples are considered to be soil samples for this screening process. As discussed in the *Final Scope of Work for the Waste Area Group 9 comprehensive Remedial Investigation/Feasibility Study at the Idaho National Engineering Laboratory* (Lee 1995), there are no complete exposure pathways for human receptors and surface water. Therefore, screening of the contaminants in the surface water is not performed. However, the analytical results for the surface water will be used to determine if the contaminants detected in the surface water were analyzed for in the sludges and subsurface soil.

5.2.2 Extent of Contamination

The nature and extent of contamination for each site was completed in Chapter 4. The nature and extent of the contamination is used to assess the physical dimensions of the contamination for each of the WAG 9

sites. The dimensions of the nature and extent of contamination will be used in the modeling of the contaminants to the ground water, human health risk assessment, and ecological risk assessment. ANL-W used the actual length and width of the ditches along with maximum depth of any contaminant above background to define the extent of contamination for each site. Almost exclusively, this conservative assumption caused ANL-W to use the maximum depth of soil above basalt in each of the release sites. The summary of the extent of contamination for the WAG 9 sites is shown in Table 5-1.

Table 5-1. Nature and extent of contamination for WAG 9 sites.

Release site	Site name	Width (ft)	Length (ft)	Depth (ft)
ANL-61A	Transformer Yard	15	21	4–9
ANL-08	EBR-II Leach Pit	18	40	0.01[a]
ANL-01	Industrial Waste Pond	200	250	0.5
ANL-01	Ditch A	5	400	0.5
ANL-01	Ditch B	5	1,400	1.3
ANL-01	Ditch C	5	500	2.5
ANL-01A	Main Cooling Tower Blowdown Ditch	6	700	2
ANL-09	Interceptor Canal	30	1,425	6
ANL-09	Interceptor Canal (Mound)	20	500	4
ANL-35	Industrial Waste Lift Station Discharge Ditch	4	500	1
ANL-53	Riser Pits (4 Pits, each with these dimentions)	4	4	1.25
ANL-53	Riser Pits (North Discharge)	6	10	1.25
ANL-53	Riser Pits (South Discharge)	6	10	1.25

a Thickness of remaining sludge in Leach Pit.

5.2.3 Separation of the Data Set by Depth

The data presented in Appendix A have been sorted into the appropriate depth ranges as indicated above for each release site. The data in Appendix A was used to prepare the screening tables in chapter 3 of this report. After sorting the data for the release sites by depth, the maximum contaminant data were screened against background. The contaminants that had concentrations less than the INEEL background were eliminated as COPCs. The contaminants that were retained indicate that at least one concentration of a COPC was detected above the background values in the depth interval shown, not that the COPC contamination extends to the bottom of the interval. For example, chromium could have a calculated 0–10 ft concentration at a given site even if the site's chromium contamination only extends from 0–4 ft. In this case, the 0–10 ft average chromium concentration shown in the table would be equal to the site's 0–4 ft average concentration. Table B-3 shows the concentration of contaminant per release site by depth used in the WAG 9 risk assessment.

The depths of contamination evaluated for the exposure routes discussed in the following sections are based on guidance given in the INEL Track-2 manual (DOE-ID 1994). Specifically, contaminant concentrations are based on the 95% upper confidence level (UCL) concentration (or maximum concentration if the maximum is less than the 95% UCL) of samples collected over the following depth ranges:

Depth	Exposure route(s)
0–6 in.	Occupational scenario: soil ingestion, inhalation of fugitive dust, and inhalation of volatiles. Note that if a composite soil sample was collected for a depth range of 0–45 cm (0–4 ft), the sample was included in the 0–15 cm (0–6 in.) data set and 0–45 cm (0–4 ft) data set.
0–4 ft	Occupational scenario: external radiation exposure.
0–10 ft	Residential scenario: all soil pathway and air pathway exposure routes.
All sample results included, regardless of depth	Residential scenario: all groundwater pathway exposure routes.

5.2.4 Calculation of the Concentration Term

For the calculation of the concentration term, the reasonable maximum exposure (RME), which is defined as the highest exposure that is reasonably expected to occur at a site (EPA 1989a), was calculated for each contaminant at each release site. A part of this RME is that the COPCs must be present at concentrations that pose a potential threat to human health. The term potential means "a reasonable chance of occurrence within the context of the reasonable maximum exposure scenario" (EPA 1989a). Because of the uncertainty associated with estimating the true average concentration at a site, the 95% UCL of the arithmetic mean is used. This value provides 95% confidence that the true site average is not underestimated (EPA 1992b).

In order to use the appropriate form of the equation to calculate the UCL, the distribution of the COPCs needs to be determined. In accordance to the guidance, the distribution was assumed to be lognormally distributed for all WAG 9 release sites. This is consistent with EPA guidance (1992b) that states "...most large or (complete) environmental contaminant data sets from soil sampling are lognormally distributed rather than normally distributed...it is reasonable to assume that Superfund soil sampling data are lognormally distributed." Thus, the UCLs for these COPCs were calculated using the following equation:

$$UCL = e^{(\bar{x}+0.5s^2+sH/\sqrt{n-1})} \qquad (5\text{-}1)$$

where

UCL = 95% upper confidence limit (mg/kg or pCi/g)

$\bar{x}$ = mean of the transformed data (mg/kg or pCi/g)

s = standard deviation of the transformed data

H = H-statistic (Gilbert 1987) one-tailed 95% value (upper bound)

n = number of samples.

The data set used in the calculation of the UCL was changed if the data had validation qualifiers of either a "U", "UJ", or "R". This means all data qualified with an "R" (reject) was removed from the sample set. For all data qualified with either the "U" or "UJ" (non-detect) qualifiers, only one-half of the reported detection limit was used in the calculation of the UCL value.

Statistics of COCs by release site are the calculated statistics for each release site and pathway (depth) are found in Appendix A. The tables show the contaminant, calculated UCL, calculated statistical average, maximum, number of samples in the release site, INEEL 95%/95% background value, and value to be used in the risk assessment. The value to be used in risk assessment was the smaller of the calculated UCL or the maximum value. On rare occasion, a COPC was eliminated from further risk assessment evaluation for a site if the calculated UCL was smaller than the INEEL 95%/95% value. This was done because in CERCLA, we are looking at the incremental increase of risk above background, and if the UCL is less than background, there is no increase in incremental risk.

In the exposure point concentration calculations, the only form of contaminant decay considered is radioactive decay (i.e., nonradionuclides are assumed to persist indefinitely in the environment). Radioactive decay is accounted for by decaying all radionuclide concentrations to July 1996 concentrations (i.e., WAG 9 Work Plan). Future radioactive decay is accounted for by decaying all radionuclide concentrations to the start of a given exposure scenario, and then calculating the average concentrations that will exist during the length of the scenario. For example, the concentration of a given radionuclide analyzed in the current occupational exposure scenario is the average concentration that would exist between 0 and 25 years in the future, and the concentration analyzed in the 100-year future residential scenario is the average concentration that would exist from 100 to 130 years.

The effects of radioactive progeny are only considered by using "+D" SFs in the radionuclide risk calculations (see Section 5.9.1.2). Decay and ingrowth calculations are not performed for complete radionuclide decay chains. The use of "+D" SFs accounts for risks produced by daughter products that are in secular equilibrium with their parent radionuclides (EPA 1994a).

While most radionuclides are more radiotoxic than chemically toxic (i.e., adverse health effects are more likely caused by the energy released during radioactive decay of the radionuclide), uranium is more chemically toxic. Therefore, to evaluate uranium for noncarcinogenic effects, the units of the radioactive uranium soil concentrations (picocuries per gram) were converted to reflect the units of nonradionuclide COPCs in soil (milligrams per kilogram) using the following equation:

$$C_s = \left(\frac{C_{sr} \times T_{1/2} \times CF_1 \times CF_2 \times CF_3}{\ln 2} \right) \times MW \times CF_4 \times CF_5 \qquad (5\text{-}2)$$

where

C_s = soil concentration of uranium in mg/kg

C_{sr} = soil concentration of the uranium isotope in pCi/g

$T_{1/2}$ = half-life of the uranium isotope in years (4.47E+09 yr for U-238)

CF_1 = conversion factor (1E−12 Ci/pCi)

CF_2 = 3.7E+10 disintegrations/Ci-s

CF_3 = 3.15E+07 s/yr

MW = molecular weight of the uranium isotope in grams per mole (238 g/mole for U-238)

CF_4 = 1 mole/6.02E+23 atoms

CF_5 = 1E+06 mg/kg.

Using this equation, the maximum concentration of only U-238 was used as the concentration term for uranium because it converts to the highest nonradioactive soil concentration by more than one order of magnitude.

5.3 Exposure Assessment

The human health exposure assessment quantifies the receptor intake of COPCs for select pathways. The assessment consists of estimating the magnitude, frequency, duration, and exposure route of chemicals to humans. To quantify the receptor intake, the following activities are performed as part of the BRA:

- Identification and characterization of exposed populations
- Evaluation of exposure pathways
- Estimation of contaminant concentrations at points of exposure
  - Soil Pathway
  - Air Pathway
  - Groundwater Pathway
- Estimation of contaminant intakes.

Each of these activities is discussed in the following sections.

5.3.1 Identification and Characterization of Exposed Populations

The following current human populations could potentially be exposed to contaminants found at or originating from WAG 9:

- Workers—Since WAG 9 is currently operational, workers at the site are potential receptors. The following two occupational exposure scenarios are analyzed in the OU 9-4 BRA:

  1. A current occupational scenario that starts now and lasts for 25 years from the present.

  2. A future occupational scenario that starts in 30 years and lasts for 25 years.

  This second occupational exposure scenario will only be evaluated if the risks from exposure to radionuclides exceed the lower limit of the NCP target risk range (i.e., 10^{-6}).

- Residents—For the purposes of the BRA, residential development will be considered as a potential future use of the site, and future residential exposure scenarios are quantitatively evaluated in the BRA. Since the nearest single-family residence is located several miles from the boundary of WAG 9, current residents will not be evaluated in the OU 9-04 BRA.

  For the purposes of the BRA, the assumption will be made that future residents will construct 10-ft basements beneath their homes. This analysis method will hereafter be referred to as a residential intrusion scenario, and all residential exposure scenario analysis in the OU 9-04 BRA will include the residential intrusion assumption. The future resident intrusion scenario will not be calculated for two sites at WAG 9. These two sites are the ANL-01-Industrial Waste Pond and the ANL-09-Canal, which are part of the natural surface runoff area for approximately 14 square miles area south of the ANL-W facility. This was based on the fact that they will always be intermittent streams and no future resident will build a house in an intermittent stream channel. As a result, all contamination detected in the alluvial material above the basalt to the 10-ft depth for each release site will be evaluated for surface pathway exposures. The depth of the soil removed to construct the basement will be the depth of alluvial material to basalt to a maximum depth of 10-ft. The soil that is removed to construct a basement will then be spread out over the entire residential lot.

The following residential scenarios will be evaluated in the OU 9-04 BRA:

1. A future residential scenario that starts in 100 years and lasts for 30 years. ANL-W is not expected to be released for residential development any sooner than 100 years in the future (DOE-ID, 1995).

2. A future residential scenario that starts in 1,000 years and lasts for 30 years.

   For nonradionuclides, chemical decay processes (e.g., microbial degradation) are not included in the baseline risk assessment. Therefore, the exposure point concentration for all exposure pathways except groundwater ingestion will remain constant for the different time-frames of interest for the residential exposure pathways. Therefore, risks and hazard quotients from exposure to the nonradionuclides will only be recalculated for the groundwater ingestion exposure point concentrations. Since the nonradionuclide concentrations will remain the same for both future residential exposure scenarios and subsequently, so will the risks and hazard quotients calculated in the 100-year future residential exposure scenario for all nongroundwater exposure pathways. But, the groundwater ingestion will be presented with the risks and hazard quotients in the 100 and 1,000-year future residential exposure scenario.Groundwater pathway risks are calculated at each 100 and 1,000-year future residential exposure scenario and are also calculated at the time of the COPCs maximum

groundwater concentration, as long as the maximum concentration occurs before 10,000 years in the future. See Section 5.7 and 5.8 for further discussion on contaminant inventory and screening and results, respectively.

5.3.2 Exposure Scenarios, Pathways, and Time Periods

Once potentially exposed populations have been identified and characterized, exposure pathways can be traced from the site to the exposed populations. Each exposure pathway describes a mechanism by which a population or individual could be exposed to contaminants originating from one or more release sites at WAG 9. Only those exposure pathways deemed to be complete (i.e., where a plausible route of exposure can be demonstrated from the site to the receptor) will be quantitatively evaluated in the BRA. The BRA risk results will be based on estimates of RME to the site's contaminants.

Based on information presented in the WAG 9 CSM (Figure 5-1 and 5-2), the following exposure scenarios, exposure pathways, and exposure routes will be evaluated in the OU 9-04 BRA:

- Exposure Scenarios
  - Occupational
  - Residential intrusion.
- Exposure Pathways
  - Groundwater
  - Air
  - Soil.
- Exposure Routes
  - Soil ingestion
  - Inhalation of fugitive dust
  - Inhalation of volatiles
  - External radiation exposure
  - Dermal absorption from soil
  - Groundwater ingestion (residential scenario only)
  - Ingestion of home grown produce (residential scenario only)
  - Dermal absorption of contaminants in groundwater (residential scenario only)
  - Inhalation of volatiles from indoor use of groundwater (residential scenario only).

5.3.3 COPC Transport

At WAG 9, the transporting pathways are:

- Soil transport pathway
- Air transport pathway
- Groundwater pathway.

Each of these pathways can be further subdivided into specific exposure routes. To estimate the exposure to hypothetical human receptors, contaminant-specific intakes were calculated for each exposure pathway. The methodology that discusses the transport of the COPC to each of the exposure routes is discussed in Sections 5.3.3.1 through 5.3.3.3. In addition, the equation used to calculate the receptor intake for each of the exposure routes is presented.

The methods used to evaluate human health exposure follow EPA guidance (EPA 1989a). Exposure parameters are obtained from standard default exposure factors guidance and EPA Region 10 guidance (EPA 1991a; 1991c). The exposure parameters used in the BRA evaluation are presented in Table 5-2. Site specific exposure parameters that will be used to calculate the realistic WAG 9 risks vary from those used in the BRA. The realistic WAG 9 exposure parameters and assumptions will be presented in the uncertainty section and used to quantify the site-specific risks with those of the BRA. Intakes were calculated using equations that include variables for exposure concentrations in the medium of concern (e.g., soil) and exposure pathway-specific parameters describing the magnitude, frequency, and duration of exposure.

To calculate the intake of arsenic, beryllium, cadmium, and hexavalent chromium from fugitive dust inhalation, the intake equation was modified to remove the intake rate (i.e., 20 m^3/d), and the body weight. This modification was necessary to use the proper toxicity values described in the following sections.

5.3.3.1 Soil Transport Pathway Methodology. Soil pathway risks are evaluated for each retained site. The following exposure routes are evaluated in the soil pathway analysis:

- Soil ingestion
- Ingestion of homegrown produce (residential scenario only)
- External radiation exposure
- Dermal absorption from soil [exposure route is insignificant for all COPCs and is not quantitatively evaluated in the BRA (see below)].

Since exposures through the soil pathway are not likely to occur from more than one release site at a time, the soil pathway is evaluated on a site-by-site basis. The possible exception to this rule is associated with the external radiation exposure route. Retained sites that have radionuclide contamination are evaluated to determine if radiation produced by one site could affect a receptor located at an adjacent site (Section 5.3.3.1.3).

As with the groundwater and air pathways, soil pathway risks and hazard quotients are calculated at 0 and 30 years in the future for the occupational exposure scenario, and at 100 and 1,000 years for the residential exposure scenario.

Table 5-2. Exposure parameters used in the OU 9-04 BRA exposure assessment.[a]

Exposure Pathway	Exposure Scenario	Intake Rate	Exposure Frequency (d/yr)	Exposure Duration (yr)	Body Weight (kg)
Soil ingestion	Occupational	50 mg/d	250	25	70
	Residential	100 mg/d (adult) 200 mg/d (child)	350	24 (adult 6 (child)	70 15
Fugitive dust inhalation	Occupational	20 m^3/d	250	25	70
	Residential	20 m^3/d	350	30	70
External	Occupational	8 h/d[b]	250	25	NA[c]
	Residential	24 h/d[b]	350	30	NA[c]
Groundwater ingestion	Residential	2 L/d	350	30	70
Food crops ingestion	Residential	0.458 g/kg-d[d] (protected fruits and vegetables and root vegetables	350	30	70
		0.343 g/kg-d[d] (exposed fruits and vegetables)			

a. Exposure parameters obtained from EPA (1991c).

b. From DOE (1994b).

c. Not applicable.

d. From EPA (1995a).

5.3.3.1.1 Transport for the Soil Ingestion Exposure Pathway—The ingestion of soil exposure route includes the evaluation of those individuals that would be exposed to the COPC in the soil. At WAG 9, this would include the evaluation of both the current and future occupational along with the future residential scenario. The ingestion of soil will use only the 70-kg adult body weight for each of the occupational scenarios and the 70-kg adult and 16-kg child body weight for the future residential scenario. The EPA makes the assumption that the mass of soil ingested by a child will be greater for a child playing in the soil than for an adult. The following equation taken directly from RAGS, shows the intake calculation for the soil ingestion pathway:

$$\text{Intake}(\text{mg}/\text{kg}-\text{day}) = \frac{\text{CS} \times \text{IR} \times \text{CF} \times \text{FI} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \qquad (5\text{-}3)$$

where

Intake = metal intake (ingestion or inhalation) (mg/kg-d)

CS = concentration of the metal (medium-specific):

- soil (mg/kg)
- air (mg/m^3)

IR = 200 mg/day for children 1 to 6 years old

= 100 mg/day age groups greater than 6 years old

CF = conversion factor (10^{-6} kg/mg)

FI = pathway specific value consideration for location

EF = 365 d/yr

ED = exposure duration (years)

BW = body weight (kg)

AT = averaging time (d); ED × 365 d/yr for noncarcinogens.

5.3.3.1.2 Transport for the Food Crop Ingestion Exposure Pathway—The ingestion of homegrown produce exposure route includes an evaluation of COPC concentrations in plants due to both root uptake and irrigation with contaminated groundwater. At each retained site, the total source concentration evaluated in the ingestion of homegrown produce exposure route is calculated by summing the 95% UCL concentration on the mean for a given COPC (or the maximum concentration if the maximum is less than the 95% UCL) with the soil concentration that would result from equilibrium partitioning between soil and groundwater contaminated with the COPC. For example, if a COPC has a concentration of 10 mg/kg at a release site, and the soil concentration of the COPC that would result from equilibrium partitioning with contaminated groundwater is 5 mg/kg, the total contaminant concentration that would be evaluated for the homegrown produce exposure route is 15 mg/kg. The effects of contaminant leaching (i.e., removal of contamination from the source zone) due to groundwater infiltration through the source zone is ignored in this evaluation.

Homegrown produce concentrations assumed for each COPC are presented in Table B-5. To evaluate the average soil concentration of radioactive COPCs in soil when irrigating with groundwater, the integrated form of Equation 5.39 in Nuclear Regulatory Commission (NRC) (1993) is used:

$$C_s(t) = \left(\frac{\frac{\dot{I}_v}{L_i + l}\left(t_e + \frac{e^{-(L_i+l)t_e}}{L_i + l}\right) + \frac{C_{so}}{L_i + l}(1 - e^{-(L_i+l)t_e}) - \frac{\dot{I}_v}{(L_i + l)^2}}{t_e} \right) \quad (5\text{-}4)$$

where

$C_s(t)$ = the average concentration of a COPC in soil for the exposure period, t_e (pCi/g)

$\dot{I}_v$ = COPC input rate from irrigation (pCi/g-d)

L_i = leach rate constant $(d)^{-1}$

λ = radioactive decay rate constant $(d)^{-1}$

t_e = exposure period [10,950 d (30 y × 365 d/y)]

C_{so} = average concentration of COPC in the top 10 ft of soil at the start of the residential exposure period (pCi/g).

For nonradioactive COPCs, this equation reduces to:

$$C_s(t) = \left(\frac{\frac{\dot{I}_v}{L_i}\left(t_e + \frac{e^{-(L_i t_e)}}{L_i}\right) + \frac{C_{so}}{L_i}(1 - e^{-(L_i t_e)}) - \frac{\dot{I}_v}{L_i^2}}{t_e} \right) \qquad 5\text{-}5$$

The COPC input rate from irrigation is given by the following equation:

$$\dot{I}_v = C_w \times \frac{I_R}{\rho \times T} \qquad (5\text{-}5)$$

where

$\dot{I}_v$ = COPC input rate from irrigation (mg/kg-d or pCi/g-d)

C_W = average concentration of a COPC in groundwater for the exposure period (mg/L, pCi/L)

I_R = irrigation rate (8.47 L/m^2-d × 90 d / 365 d) (Maheras et al. 1994)

ρ = soil density (1.5E+06 g/m^3)

T = assumed thickness of root zone (0.2 m) (IAEA 1994).

The leach rate constant is given by the following equation (Baes et al. 1983):

$$L_i = \frac{P}{\theta_c \times \left(1 + \frac{K_d \times \rho}{\theta_c}\right) \times T} \times CF \qquad (5\text{-}6)$$

where

P = net water percolation rate (0.36 m/y) [infiltration rate of 0.1 m/y as presented in DOE (1994) plus the contribution from irrigation]

θ_c = volumetric water content in source volume (0.41 m^3/m^3) (Rood 1994)

K_d = COPC-specific soil-to-water partition coefficient (cm^3/g)

ρ = soil density (1.5 g/cm^3)

T = assumed thickness of root zone (0.2 m) (IAEA 1994)

CF = conversion factor (1 y/365 d).

The radioactive decay constant is given by the following equation:

$$\lambda = \frac{\ln 2}{T_{1/2}} \tag{5-7}$$

where

$T_{1/2}$ = the half-life of a radionuclide (d).

5.3.3.1.3 Transport for the External Exposure Pathway. For the external radiation exposure route, standard EPA protocols are used to estimate risks for all retained sites. In other words, external radiation exposure risks are calculated by multiplying radiation intakes for specific isotopes by the radionuclide slope factors presented in EPA's Health Effects Assessment Summary Tables (HEAST) (EPA 1994a) as shown in Table B-10. The standard EPA protocols are used because most of the retained sites in the BRA have radionuclide contamination that is at least 6 in. thick over a relatively large area. This thickness is large enough to satisfy the assumption that an increase in source thickness will not cause an increase in surface radiation exposures.

5.3.3.1.4 Transport for the Dermal Contact with Soil Exposure Pathway. For the dermal absorption from soils exposure route, EPA (1992b) shows that if the absorption fraction for a given COPC is less than 0.1, then the dermal absorption from soils exposure route produces risks that are less than risks produced by the ingestion of soil exposure route for that COPC. The equation for calculating soil dermal absorption fractions reported in EPA (1992b) is shown below. This equation is currently under review by the EPA Office of Research and Development because of inconsistencies in actual inorganic absorbed fractions compared to inorganic absorbed fractions predicted by the equation.

$$ABS = \frac{\rho_{soil} K^{soil}}{AF(k_{soil} + k_{vol})}\left[1 - e^{-(k_{soil} + k_{vol})t_{event}}\right] \tag{5-8}$$

where

ABS = absorption factor for dermal contact with soil (unitless)

ρ = density of soil (g/cm^3)

K^{soil} = skin permeability coefficient for chemicals in soil (cm/hr)

AF = soil to skin adherence factor (mg/cm^2)

k_{soil} = $K^{soil}\ \rho_{soil}\ 1{,}000/AF\ (hr^{-1})$

k_{vol} = $K_h\ D_{air}\ 3{,}600/(AF\ K_D\ l)\ (hr^{-1})$

t_{event} = time (hr).

and

$$K^{soil} = \frac{K^{water}}{k_{soil} / w} \qquad (5\text{-}9)$$

where

K^{water} = skin permeability coefficient for chemicals in water (cm/hr)

$K_{soil/w}$ = soil/water partition coefficient (unitless) = $K_D\ \rho_{soil}$.

Absorption fractions calculated using the above equations are presented in Table B-1, column 3. As shown in the table, absorption fractions for all WAG 9 COPCs are less than or equal to 1E–03, which is a value that is one hundred times lower than the screening limit identified in EPA (1992b); therefore, the dermal absorption from soil exposure route is not evaluated further in the BRA.

ANL-W used the EPA Region III guidance for Assessing Dermal Exposure from Soil (EPA 1995) to assess the dermal exposure at ANL-01-Ditch A. This ditch was selected because it has four contaminants listed on the EPA region III guidance. The calculated dermal exposure pathway values for this ditch were calculated and compared to the risks for the soil ingestion pathway. The comparison in risks are shown in Table B-34 of Appendix B. ANL-W used the same default parameters of exposed skin area (hands and arms 0.312 m^2) and adherence factor (0.95 based on soil size) that were used at WAG 8. The results show that for methylene chloride, chloroform, and Bis (2-ethylhexyl)phthalate the risks for the soil ingestion pathway are greater than those calculated for the dermal. But, for arsenic the calculated risk for the dermal is 1E-04 using the EPA region III guidance while the soil ingestion risk is only 7E-05. This increase in dermal risk for the arsenic is not surprising. The IDHW risk assessor stated that other WAGs have noticed the same trend. ANL-W has not completed the dermal exposure for any of the other release sites.

5.3.3.2 Air Transport Pathway Methodology. All sites that have contamination in the top 10 ft of soil are assumed to have a contaminant source that can be released into the air pathway. Again, only the alluvial material can be transported in the air; only the actual depth of soil above the basalt to a maximum depth of 10 ft will be used in the WAG 9 BRA. The exposure routes that are evaluated as part of the air pathway analysis are:

- Inhalation of fugitive dust
- Inhalation of volatiles.

Since there is a possibility that contamination from multiple sites can mix together within the air volume above WAG 9, the air pathway is analyzed in a cumulative manner in the OU 9-04 BRA. In order to perform this cumulative analysis, a WAG-wide average soil concentration is calculated for each COPC. The concentration of each COPC in the respirable particulate matter above the WAG is assumed to equal

this average soil concentration. Averaging contaminant concentrations over the WAG for the air pathway produces one contaminant-specific risk estimate for each air pathway exposure route (i.e., for each time period, each air pathway exposure route has the same risk or HI at every retained site).

5.3.3.2.1 Transport for the Fugitive dust Inhalation Exposure Pathway—The equations discussed below are used to estimate airborne contaminant concentrations:

$$C_{air} = CF\,1\times10^{-6}\,R\,C_{soil} \qquad (5\text{-}10)$$

where

C_{air} = contaminant concentration in air (mg/m^3 or pCi/m^3)

CF = conversion from kg to mg for nonradionuclides or g to mg for radionuclides

R = airborne respirable particulate matter concentration (0.011 mg/m^3) (Mitchell et al. 1995)

C_{soil} = WAG average contaminant soil concentration (mg/kg or pCi/g) weighted by site area

and

$$C_{soil} = \frac{\sum C_n A_n}{A_T} \qquad (5\text{-}11)$$

where

C_n = contaminant soil concentration at site *n* (mg/kg or pCi/g)

A_n = surface area of site *n* (m^2)

A_T = total area of the WAG 9 retained sites (m^2)

n = number of sites.

5.3.3.2.2 Transport for the VOC Inhalation Exposure Pathway—The equation used for estimating concentrations of airborne volatiles is:

$$C_{soil} = \frac{\sum (C_n / VF_n) A_n}{A_T} \qquad (5\text{-}12)$$

where

C_n = contaminant soil concentration at site *n* (mg/kg)

VF_n = volatilization factor [as described in INEL Track 2 guidance (DOE-ID 1994)] for site *n* (m^3/kg)

A_n = surface area of site *n* (m^2)

A_T = total area of the WAG 9 retained sites (m^2).

These equations produce conservatively high estimates of airborne COPC concentrations because no credit is taken for dilution of airborne concentrations due to dust blown from uncontaminated areas of WAG 9.

As with the soil pathway analysis, the air pathway receptor is either a current occupational worker (who is assumed to be exposed for 25 years) or a hypothetical future resident (who is exposed for 30 years). Air pathway risks and hazard quotients are calculated at 0 and 30 years in the future for the occupational scenario, and at 100 and 1,000 years in the future for the residential scenario. Estimated concentrations of COPCs in fugitive dust for each exposure period are presented in Table B-6, and estimated concentrations of volatiles for each time period are presented in Table B-7.

5.3.3.3 Groundwater Transport Pathway Methodology. To quantify risks for the future residential receptor (there is no occupational receptor for this exposure pathway), modeling of contaminant concentrations in groundwater is required. The conceptual model for groundwater transport is presented in Section 5.5. This Section only presents the calculations for the groundwater transport pathway methodology. The exposure routes are evaluated as part of the WAG 9 groundwater pathway analysis:

- Ingestion of groundwater
- Dermal absorption of groundwater
- Inhalation of volatiles produced by indoor use of groundwater.

Exposures to COPCs through dermal absorption of groundwater are controlled by a given COPCs permeability coefficient of water through skin (K_p^W) According to EPA (1992b), if the permeability coefficient for a given COPC is less than 0.1 cm/hr, then the dermal absorption from groundwater exposure route produces risks that are less than risks produced by the ingestion of groundwater exposure route for that COPC. In the BRA, the default permeability coefficient used for inorganic COPCs is 1E-03 cm/hr, and the permeability coefficients for organic COPCs are estimated using the equation (EPA 1992b);

$$Log\ K_p^w = -2.72 + 0.71\ Log\ K_{ow}\ 0.0061\ MW \qquad (5\text{-}13)$$

where

K_{OW} = octanol/water partition coefficient (unitless)

MW = molecular weight (g/mol).

Permeability coefficients for WAG 9 COPCs are shown in Table B-1, Column 4. Since many of the organics have permeability coefficients that are greater than the 0.1 cm/hr screening level, the dermal absorption from groundwater exposure route is quantitatively evaluated in the BRA. Contaminant intakes for this exposure route are calculated using the equations shown in Section 5.3.3.4.

In the BRA, exposures due to inhalation of water vapors from indoor water use are calculated based on experimental data presented in Andelman (1990). This study derived a volatilization constant that defines the relationship between the concentration of a contaminant in household water and the average concentration of the volatilized contaminant in air. In the derivation, all uses of household water were considered (e.g., showering, laundering, dish washing), and certain reasonable assumptions were made in deriving a volatilization fraction. For example, the study included assumptions about water usage for a family of four, the volume of the dwelling, and the air exchange rate. Furthermore, the study assumed that the average transfer efficiency weighted by the type of water use is 50% (i.e., half of the concentration of each chemical in water will be transferred into air by all types of water uses).

In the BRA indoor water use analysis, a central tendency value for a COPCs volatilization fraction [6.50E-02 mg/m^3 air per mg/L water (Andelman 1990)] is used to develop estimates of COPC airborne concentrations. The airborne concentrations are calculated by multiplying the central tendency value by the COPC groundwater concentrations shown in Tables 5-26 through 5-29. These concentrations are then used to develop contaminant intake estimates using the equations shown in Section 5.3.3.4. The estimates of COPC airborne concentrations from indoor water use calculated for the BRA are shown in Table B-8.

5.3.3.4 Estimation of Contaminant Intakes. The general equation that is used to calculate intakes for most of the OU 9-04 BRA exposure routes is (EPA 1989b);

$$Intake = \frac{C \times IR \times EF \times ED}{BW \times AT} \qquad (5\text{-}14)$$

where

Intake = contaminant intake (mg/kg-d)

C = concentration of a given contaminant in a contaminated media (e.g., soil, air, water, etc.) (mg/kg, mg/m^3, mg/L, etc.)

IR = ingestion rate of the contaminated media (mg/day, m^3/day, L/day, etc.)

EF = exposure frequency (day/yr)

ED = exposure duration (yr)

BW = body weight (kg)

AT = averaging time (yr).

The above equation applies to all exposure routes except exposure to external radiation. For the external radiation exposure route, intakes are calculated using the following general equation:

$$Intake = C \times ET \times EF \times ED \times CF \qquad (5\text{-}15)$$

where

Intake = radiation intake (pCi-yr/g)

C = radionuclide concentration in soil (pCi/g)

ET = exposure time (hr/d)

EF = exposure frequency (day/yr)

ED = Exposure duration (yr)

CF = Conversion factor (1.14E-04 yr/hr).

The specific intake factor equations used for the BRA exposure routes are shown in Tables B-9 through B-19. Results of the BRA intake factor calculations are shown in Table B-20 through B-24.

5.4 Groundwater Transport Modeling

5.4.1 Review of Track 2 Groundwater Modeling

Prior to this comprehensive RI/FS baseline risk assessment effort, sites currently retained in the baseline risk assessment (BRA) had been examined in Track 1 investigations, some were re-examined in follow-up Track 2 reports, while some were documented in Preliminary Scoping Packages (PSP) for Track 2 investigations. The Track 2 and PSP reports generally contain results of prior groundwater pathway risk analyses. These include groundwater ingestion risk estimates for future residents calculated with the use of the groundwater pathway screening code, GWSCREEN (Rood 1994). The dates of these earlier investigations indicate the use of previous, older versions of the code, some of which were limited in the types of modeling allowed (i.e., earlier versions did not contain a pond model option or vertical dispersion).

The results of these earlier investigations of groundwater risks led to the inclusion of sites in the final BRA work. Details of the earlier investigations are summarized in Table 5 of Appendix J. That information is provided in this report to allow comparison of what had previously been done to the modeling work developed for the final BRA. Important modeling assumptions and parameter choices resulted in a different range of groundwater ingestion risks.

Surface contamination sites included in this analysis of groundwater ingestion risks are ANL-01 (Industrial Waste Pond and Ditches A, B, and C), ANL-01A (Main Cooling Tower Blowdown Ditch), ANL-08 (EBR-II Leach Pit), ANL-09 (Interceptor Canal), ANL-35 (Industrial Waste Lift Station Discharge Ditch, also known as the North Ditch), ANL-53 (Cooling Tower Riser Pits and discharge areas), and ANL-61A (PCB-contaminated soil adjacent to ANL-61). In general, these previous analyses used infiltration rates of 0.1 m/yr, though some used much higher values. Unsaturated zone thickness was typically 19.2 m but some sites were modeled with very different values. The compounds previously analyzed at each site and their resulting groundwater ingestion risk numbers are discussed below.

5.4.1.1 ANL-01 (Industrial Waste Pond and Ditches A, B, and C). The Industrial Waste Pond (IWP) and associated ditches (Ditches A, B, and C) were analyzed for groundwater ingestion risks in a 1995 Revised Preliminary Scoping Package (ANL-W 1995). That document presents the results of two separate analyses. The first was conducted with the concentration results of soil sampling events performed in 1986 and 1989. The second analysis was based on more recent sampling (1994).

In the first analysis, beryllium, chromium, mercury, silver, and the pesticide Silvex were examined. Forward calculations based on maximum detected concentrations in soil samples collected from the three ditches were performed for the following contaminants (and their concentrations): Be (3.9 mg/kg), Cr-III (1,150 mg/kg), and Hg (1.4 mg/kg). Forward risk calculations for the IWP sediments included the compounds (and their maximum detected concentrations in the IWP): Cr-III (9,900 mg/kg), silver (6.6 mg/kg), and Silvex (27.6 mg/kg). Hexavalent chromium for soils was analyzed using SW-846 method 7196A on the extract that was leached from the soil using SW-846 method 1310. Chromium was modeled only as trivalent chromium since no hexavalent chromium was detected in any of the samples. Beryllium was analyzed for both carcinogenic and noncarcinogenic effects. The results for the ditch analysis indicated a residential groundwater ingestion risk of 1.24E–6 for Be. The calculated residential groundwater ingestion hazard quotients associated with the ditches were 1.6E–05 (Be), 3.3E–03 (Cr-III), and 4.4E–03 (Hg). The calculated residential groundwater ingestion hazard quotients associated with the IWP sediments were 5.3E–06 (Ag), 2.0E–5 (Cr-III), and 8.7E–02 (Silvex). This analysis used 1.0 m/yr for the infiltration rate for some contaminant runs but 6.9 m/y was also used.

An additional contaminant, Th-230, was modeled for groundwater ingestion risks based on annual IWP water samples. Th-230 was chosen because the isotope has the highest slope factor of the radionuclides detected in the pond water. The GWSCREEN results indicated a future residential groundwater ingestion risk of 6.47E–20.

The more recent analysis involved the use of the maximum detected contaminant concentrations from 1994 soil samples. In that analysis, backward calculations produced risk-based soil concentrations for antimony, arsenic, cadmium, chromium, cyanide, lead, mercury, nickel, selenium, silver, vanadium, zinc, Cs-137, Co-60, Sr-90, U-238, and Cm-244. The analysis concluded that none of these contaminants were detected in the IWP or three ditches at concentrations that exceed risk-based concentrations.

5.4.1.2 ANL-01A (Main Cooling Tower Blowdown Ditch). The Main Cooling Tower Blowdown Ditch (MCTBD) was most recently analyzed for groundwater risks in 1993. The Revised Preliminary Scoping Package (ANL-W 1995) contains the results of both forward and backward calculations. These were prepared with GWSCREEN version 2.01 using an infiltration rate of 1.0 m/yr and the maximum sampled quantities of beryllium, chromium, mercury, and silver. Both carcinogenic and noncarcinogenic effects of beryllium were examined. Chromium was examined as trivalent (Cr-III); the documentation notes that hexavalent (Cr-VI) chromium would be reduced to trivalent chromium prior to discharge. Chromium was examined with a concentration of 386 mg/kg from 4–8 ft below grade. The other contaminants were assumed to exist at 0–4 ft below grade at concentrations (in mg/kg) of 4.2 (Be), 0.35 (Hg), and 1.7 (Ag).

Results for beryllium were a hazard quotient (HQ) of 2.92E–04 and a carcinogenic risk of 2.69E-06; the calculated beryllium cancer risks exceeded the criterion of 1E–06. The HQ values calculated for chromium (Cr-III), mercury, and silver were 3E–08, 8E–04, 3E–04, respectively.

5.4.1.3 ANL-08 (EBR-II Leach Pit). The EBR-II Leach Pit has been extensively examined in the past for groundwater impacts. Most recently, the Leach Pit was analyzed in a Preliminary Scoping Track 2 Summary Report (RUST Geotech Inc. 1994). The analysis included backward calculations of limiting soil concentrations for the 8 in. of contaminated pit sludge at the bottom of the pit prior to the 1993 removal. Follow-up forward calculations of groundwater risks were performed for the 1/16-in. contaminated sludge remaining at the bottom of the pit following sludge removal activities. The analyses considered infiltration rates of both 0.1 m/yr and 5.0 m/yr. The first value is the accepted Track 2 default value for background

infiltration conditions (DOE 1994) and was assumed for the post-removal conditions that include a bentonite cap in the pit and backfilled to grade. The 5.0 m/yr value represents the maximum estimated rate based on 1969 liquid discharge data (90,000 gal/yr) and known pit dimensions (RUST Geotech Inc. 1994).

Initially, the backward-calculated limiting soil concentration values were found for a suite of suspected Leach Pit contaminants including non-radiological types (silver, arsenic, barium, beryllium, cadmium, chromium, mercury, manganese, nickel, unidentified PCBs, lead, antimony, tin, thallium, vanadium, and zinc), radiological types (Am-241, Co-60, Cs-134, I-129, Np-237, Pu-238, Pu-239, Sr-90, U-234, U-235, U-238, and Y-90), and a host of dioxin/furan contaminants. The 5.0 m/yr infiltration rate for Leach Pit operational conditions was used in the backward calculations.

The dioxin/furan contaminants (2,3,7,8 Tetrachlorinated dibenzofuran, 1,2,3,7,8 Pentachlorinated dibenzofuran, 2,3,7,8 Pentachlorinated dibenzofuran, 2,3,7,8 Hexachlorinated dibenzofuran, 2,3,7,8 Heptachlorinated dibenzofurans, and other hexachlorinated, heptachlorinated, and octachlorinated dibenzofurans) were modeled as a TCDD-equivalent compound. Arsenic and beryllium were examined using both carcinogenic and noncarcinogenic effects data. Chromium was analyzed as both trivalent and hexavalent chromium. The short half-life (2 yr) of Cs-134 indicated this contaminant would not present unacceptable risks to a future resident via the groundwater pathway; as a result, Cs-134 was not modeled.

The resulting limiting soil concentrations were compared to the maximum observed concentrations detected in Leach Pit sludge samples. If the maximum detected concentration was greater than the GWSCREEN-modeled limiting soil concentration, the contaminant was included in the forward risk calculation; otherwise, the contaminant was not considered to pose unacceptable risks to a future resident via the groundwater pathway and was omitted from further analysis. The contaminants failing this screening process in the 1994 analysis included arsenic and beryllium (both as carcinogens), the TCDD-equivalent, the PCBs (analyzed as "total" PCBs), I-129, and Np-237.

The forward calculations were based on the post-removal conditions of 1/16th-in. remaining sludge and 0.1 m/yr infiltration rate. The unscreened contaminant inventories were based on the volume of sludge estimated to be remaining and the maximum observed concentrations. Maximum groundwater risks and groundwater concentrations between 4 and 3,470 years were calculated. Np-237 was modeled using a maximum concentration of 0.1±0.2 pCi/g for a total inventory 5.26E–05 Ci. The analysis of Np-237 included the progeny Pa-233, U-233, Th-233, Ra-225, Ac-225 and yielded a total carcinogenic future groundwater ingestion risk of 1.4E–06. I-129 was analyzed using a concentration of 0±0.9 pCi/g (for a total inventory of 1.98E–05 Ci) and yielded a maximum carcinogenic risk of 1.3E–08.

Arsenic was modeled with a concentration of 52.8 mg/kg and yielded a maximum carcinogenic risk of 3.4E–07. Beryllium was modeled with a maximum concentration of less than 2 mg/kg (1.21E+05 mg total inventory) and yielded a maximum carcinogenic risk of 3.7E–06. Total PCBs were modeled with a concentration of 7.9 mg/kg and yielded a maximum carcinogenic risk of 1.4E–08. The TCDD-equivalent was modeled with a concentration of 0.012 mg/kg and yielded a maximum risk of 6.5E–08.

Np-237 and beryllium (as a carcinogen) failed the 1E–06 criterion. In addition, the 1994 assessment did not attempt to account for any of the contamination that leached from the pit into the vadose zone and which may still pose a threat to future residents despite the sludge removal action. The evaluation of the potential vadose contamination resulting from Leach Pit operations is included in the final BRA analysis.

5.4.1.4 ANL-09 (Interceptor Canal). The Interceptor Canal was previously examined for risks associated with the groundwater pathway. The analysis is presented in a Revised Preliminary Scoping Package for ANL-09 (ANL-W 1995). The infiltration rate used in the runs was 0.1 m/yr; the unsaturated thickness was set at 63.5 m. Both forward calculation of risks for carcinogens and hazard quotients for noncarcinogens as well as backward calculations of each were prepared. For the forward calculations, the analyzed contaminants (and their maximum concentration in mg/kg or pCi/g) were: antimony (14.7), arsenic (11.9), lead (39.7), mercury (0.33), selenium (0.43), silver (1.6), cyanide (7.9), Am-241 (0.19), Cm-244 (0.09), Cs-137 (55.0), Sr-90 (6.4), Th-230 (2.4), and U-238 (2.5).

Results for the residential scenario of groundwater ingestion for the examined contaminants are given as hazard quotients: 0.08 (antimony), 1.0 (arsenic), 9E–04 (mercury), 2E–03 (selenium), 4E–04 (silver), and 4E–02 (cyanide). The radionuclide analysis returned risks of <1E–06 (Am-241, Cs-137, and Sr-90), 9E–10 (Cm-244), 7E–08 (Th-230), and 2E–07 (U-238). Lead was not analyzed due to the lack of toxicity data. Arsenic was also examined for carcinogenic risk and yielded the one result (3E–04) that exceeded the criterion of 1E–06 risk.

5.4.1.5 ANL-35 (Industrial Waste Lift Station Discharge Ditch, also known as North Ditch). The North Ditch groundwater pathway risks were previously analyzed in the Preliminary Scoping Package for ANL-35 (ANL-W 1995). Both forward calculation of risks for carcinogens and hazard quotients for noncarcinogens as well as backward calculations of each were prepared. Contaminant impact to groundwater were modeled using an infiltration rat of 1.0 m/yr and contaminant inventories based on maximum soil sample concentrations. Investigated contaminants (and their maximum concentrations in mg/kg or pCi/g) were: antimony (44.4), arsenic (10.3), barium (647), cadmium (4.8), chromium (118), copper (479), lead (47.2), manganese (1200), mercury (22), selenium (0.78), silver (352), zinc (491), cyanide (14.3), Cs-137 (2.2), and U-238 (3.1). Chromium was examined as both trivalent (Cr-III) and hexavalent (Cr-VI) forms.

Results for the residential scenario of groundwater ingestion for the examined contaminants are given as hazard quotients: 0.01 (antimony), 0.06 (arsenic), 6E–04 (barium), 0.02 (cadmium), 3E–04 (Cr-III), 7E–02 (Cr-VI), 2E–03 (copper), 0.02 (manganese), 1E–04 (mercury), 3E–04 (selenium), 7E–03 (silver), 5E–04 (zinc), and 4E–03 (cyanide). The radionuclide analysis returned risks of <1E-06 (Cs-137) and 2E–07 (U-238). Lead was not qualitatively analyzed due to the lack of toxicity data for this element. Arsenic was also examined for carcinogenic risk and yielded 1E–05.

5.4.1.6 ANL-53 (Cooling Tower Riser Pits and discharge areas). The Cooling Tower Riser Pits were examined for groundwater ingestion risks in a 1993 Track 2 Preliminary Scoping Package (ANL-W 1993). GWSCREEN was used to calculate groundwater ingestion risks using an infiltration rate of 0.1 m/yr and an unsaturated thickness of 4.7 m.

The analysis included contaminated soil associated with the riser pits and contaminated soil at the surface discharge areas. The contaminants (and the analyzed concentrations) at the riser pits included arsenic (76 mg/kg), mercury (0.78 mg/kg), chromium (1,727 mg/kg), and lead (4,725 mg/kg). These concentrations represent maximum detected concentrations from 11.5 ft below grade.

The contaminants (and the analyzed concentrations) at the surface discharge areas included arsenic (7.7 mg/kg), mercury (0.40 mg/kg), chromium (59 mg/kg), and lead (43 mg/kg). The surface discharge concentrations represent maximum values detected in the first ten inches of soil below grade. As a result of

the shallow extent of contamination, the surface discharge areas were analyzed for soil ingestion and inhalation pathways; not for groundwater impacts.

Chromium was analyzed for both the trivalent (Cr-III) and hexavalent (Cr-VI) form and only detected in the trivalent form.. The lack of toxicity data precluded the quantitative examination of lead in the forward calculations of risk. The groundwater analysis results for the remaining riser pit contaminants, as expressed as hazard quotients, were 1.6E–02 (As); 8E–06 (Hg), and 8E–06 (Cr-III).

5.4.1.7 ANL-61A (PCB-contaminated soil adjacent to ANL-61). The PCB-contaminated soil site associated with an underground fuel storage tank in the ANL-61 Transformer Yard has not been previously analyzed for groundwater impacts. Two soil samples returned concentrations of 39 and 55 mg/kg of unidentified PCBs.

5.5 Conceptual Model for Groundwater Transport

5.5.1 Model Selection

The GWSCREEN model (Rood 1994) was selected to perform the groundwater fate and transport calculations. The model was designed to perform groundwater pathway screening calculations for the Track 1 and 2 process. It is also an appropriate model to use when site characterization data are lacking and little would be gained by the use of a more complex model. Other more sophisticated models were informally reviewed but were not selected to perform groundwater pathway calculations because:

- The probability of risks significantly exceeding acceptable criteria was low and did not warrant the use of a more sophisticated model at this time in the process.
- Additional field characterization data would likely need to be obtained to fully utilize the capabilities of a more complex model.
- The results of this analysis will indicate what operable units, if any, require further attention. The use of a more complex model at such time may justified.

5.5.2 Source Areas

Source areas were modeled individually instead of modeling a single, composited site. Each release site within ANL-W was represented by rectangular areas that were oriented parallel to north-south or east-west lines (Figure 5-3). Actual dimensions used in the model site are described in Table 5-1, but were modified to account for irregular geometry and source orientation. The total volumes of each modeled source however, were made to approximate the actual estimated contaminated volumes (see Table 5-24). A total of six release sites were modeled. Release sites ANL-01 and ANL-01A required several rectangular areas (elements) to represent the source.. Six rectangular area sources were used to model ANL-01, and two area sources were used to model ANL-01A and ANL-09. Each retained site was located according to its physical geographic location within the ANL-W facility.

A receptor grid was overlain on the source areas such that contributions to contaminant concentrations from all retained sites could be calculated at each receptor node. ANL-35 served as the center of the grid. The distance from the center of ANL-35 to the center of each area source was computed

(listed in columns 4 and 5 of Table 5-3) and provided a means to relate the receptor grid to all source areas in the model domain.

Each source area was modeled either as a pond or a surficial or buried source as described in the GWSCREEN user's manual. Pond sources were assigned to retained sites that were either unlined ditches or infiltration ponds, and currently or in the past received liquid effluent. Infiltration rates were based on the amount of liquid effluent the ditches received plus the amount of rainfall runoff the ditch collects. The pond source model was used for the following retained sites: ANL-09—Canal, ANL-01—Ditch A, ANL-01—Ditch C, ANL-01—Ditch B element 2, ANL-01—Industrial Waste Pond, ANL-08—Leach Pit, and ANL-35—North Ditch.

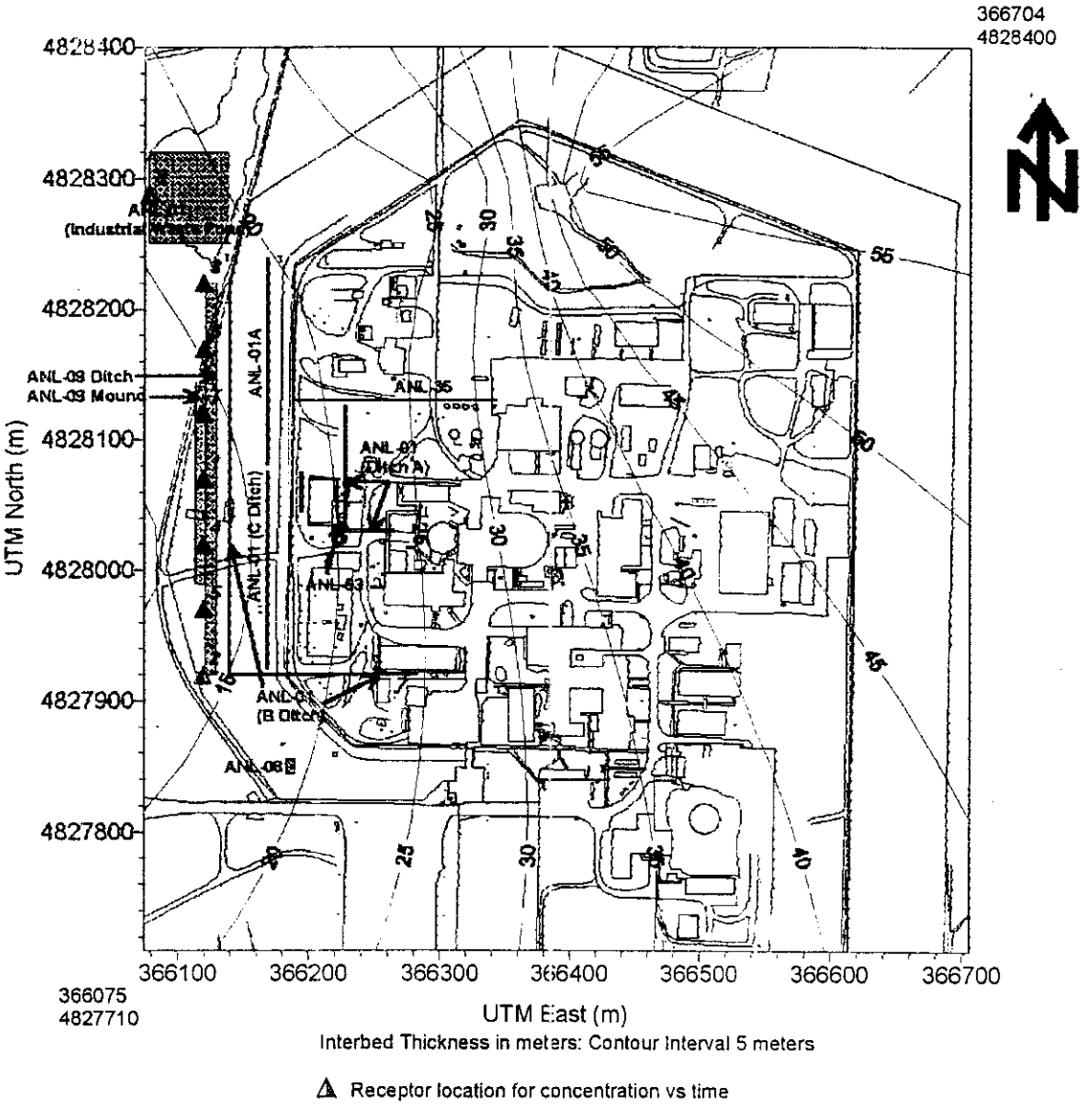

Figure 5-3. Source configuration for retained sites in WAG 9. Interbed thickness are interpolated from 9 wells in the vicinity of the site. All interbed thicknesses are the total of all interbeds from ground surface to top of aquifer surface. See Section 2.4.2 for discussion of discontinuous interbeds.

Table 5-3. Location of source areas and source area dimensions representing operable units modeled for ANL-W.

Source Element	UTM Coordinates of Source Center Easting (m)	Northing (m)	Xa (m)	Ya (m)	Length east-west (m)	Length north south (m)	Thickness (m)	Volume (m^3)
ANL 09 Canal[b]	366126	4828070	141	60	9.1	300	1.8	5020
ANL-09 Mound	366117	4828065	150	65	6.1	152	1.2	1133
ANL-01 B Ditch element 1	366140	4828070	127	60	1.5	305	0.4	185
ANL-01 B Ditch element 2	366213	4827920	54	210	140	1.5	0.4	85
Total ANL-01 B Ditch								270
ANL-01 C Ditch	366169	4828000	98	130	1.5	152	0.76	176
ANL-08 EBR-II Leach Pit	366187	4827850	80	280	6	12	0.11	7.9
ANL-01A element 1	366169	4828160	98	−30	1.8	158	0.61	176
ANL-01A element 2	366195	4828060	72	70	1.8	31	0.610	35
Total ANL-01A								211
ANL-01 Ditch A element 1	366228	4828080	39	50	1.5	91	0.60	81.9
ANL-01 Ditch A element 2	366245	4828030	22	100	41	1.5	0.60	36.9
Total ANL-01 Ditch A								119
ANL-35 North Ditch	366267	4828130	0	0	1.2	152	0.31	56.6
ANL-53 Riser Pits	366225	4828040	42	90	3.4	4	0.48	5.17
ANL-01 Industrial Waste Pond	366109	4828288	158	−158	61	76	0.15	695.4

a. X, Y Coordinates (based on ANL-35 as center).
b. Area of the ditch actively receiving run off water or liquid effluent was included in the source. The actual area of the ditch is larger. The contaminant inventory reported in Table 5-17 was assumed to be uniformly distributed in the smaller volume representing the active area of the ditch.

The surface or buried source model was assigned to all other sources. These sources included ANL-53—Riser Pits, ANL-01—Ditch B element 1, and ANL-09—Mound. ANL-01—Ditch B element 1 was not modeled as a pond source because the ditch has been filled in and no longer receives liquid effluent or storm runoff.

Surface or buried sources, and pond sources are modeled in GWSCREEN as volumes where the contaminant is assumed to be homogeneously mixed. Steady state infiltration under unit gradient conditions are assumed. Contaminants are assumed to be in solid and aqueous phase equilibrium and equilibrium concentrations are described by the linear sorption coefficienet or K_d. Leaching from the source volume to the underlying strata is assumed to be a first-order process wnere the fraction leached is constant and the mass flux is proportional to the amount of contaminant present in the source volume. Radioactive decay and contaminant degradation are considered.

5.5.3 Unsaturated Zone

The Track 1 and 2 groundwater modeling process typically only considered water travel time through sedimentary interbeds and assumes the transit time through fractured basalt is relatively

instantaneous. The Large Scale Infiltration Test in performed at the INEEL 1995 came to a similar conclusion that transit time in the unsaturated zone was controlled by the hydraulic properties of the sedimentary interbeds and not the fractured basalt. Recent modeling studies performed for Radioactive Waste Management Complex at the INEEL (Magnuson and Sondrup 1996) have also concluded that transit time is primarily controlled by the sedimentary interbeds. This approach has been incorporated into this analysis. Interbed thickness is known to vary across the INEEL and the ANL-W site. In addition, the interbeds below the ANL-W site are discontinuous as discussed in Section 2.4.2. Thus, site specific well logs have been used to delineate the interbed thickness below the retained sites (Figure 5-3). The advantage to the conceptual model employed in this analysis is that each source area may be assigned an interbed thickness that coincides with the actual estimated interbed thickness underlying the source. Using a single composite source as was originally proposed does not allow for this refinement.

The unsaturated zone in GWSCREEN is modeled using a plug flow model. Dispersion and diffusion are ignored; only radioactive decay and contaminant degradation reduce contaminant concentrations in leachate. Unit gradient conditions are assumed throughout the unsaturated zone. Contaminant travel time are governed by the water infiltration rate, contaminant sorptive properties, and the hydraulic properties of the interbeds.

5.5.4 Saturated Zone

The saturated zone (Snake River Plain Aquifer) is modeled as a homogeneous isotropic aquifer of infinite lateral extent and finite thickness. No sources or sinks are considered and a steady state uniform flow field is assumed. Contaminants enter the aquifer in an area defined by the length and width of the source area and disperse both horizontally and vertically as they are transported downgradient. Equilibrium sorption reactions described by the K_d value are included. Contaminant concentrations are evaluated by averaging the concentration in the first 15 m (measured from the surface of the aquifer) of the aquifer. The 15 m averaging concentration depth was chosen based on the default Track 2 length of well screen (DOE-ID 1994b). This closely resembles the 18.5 m average well screen length of the six ANL-W wells presented in Table 4-2. Contaminants are allowed to disperse completely in this effective aquifer thickness as they move downgradient.

Groundwater flow direction underneath ANL-W is reported to be south 60 degrees west (240 degrees) based on flow lines reported in Arnett et al. 1994. To simplify the GWSCREEN calculations, we reoriented the flow direction to be westward (270 degrees). This assumption may be nonconservative for some sites, overly conservative for others, but the overall effect may be minimal. Gradients in the Snake River Plain Aquifer underneath ANL-W are relatively shallow and the actual flow direction is not precisely known. It was suggested that flow toward the south also be considered. Changing the orientation of flow for sites that are approximately equal length on both sides will have little effect on the maximum concentrations. However, for sites that have significantly different length and width dimensions, groundwater flow direction will influence the estimated maximum concentration. Source areas such as ANL-35 generate higher groundwater concentrations when the groundwater flow is oriented east-west compared to north south. Groundwater concentration results were calculated primarily for groundwater flow in the east-west direction because:

- The estimated flow path is nearer to the east-west direction compared to the north south.
- Elongated sources areas such as ANL-09, are actually more complex, containing several points at which the source orientation changes with respect to groundwater flow.

- Groundwater flow orientation has little impact on maximum concentrations predicted for sources with approximately equal length and width

- A change in groundwater flow direction will increase predicted concentrations from some source areas while decreasing concentrations from others.

To address the possibility of north-south oriented groundwater flow, maximum groundwater concentrations were calculated for selected sites assuming flow from the north. Results are only presented as a sensitivity-uncertainty analysis case and risk values are calculated only for the east-west groundwater flow condition.

5.6 Groundwater Transport Parameters

In this section, we describe the input parameter values for the GWSCREEN model for each retained sites source elements modeled. Parameter values are provided for the source, unsaturated, and saturated zone. Contaminant sorption coefficient values (K_d) used are the same as those used in the human health risk assessment and are shown by contaminant in Appendix B.

5.6.1 Source Area Parameters

5.6.1.1 Length, Width, and Thickness of Sources. Length, width, and thickness of each modeled source area is provided in Table 5-3. Dimensions were based on site surveys and measurement data described in the Nature and Extent of Contamination, Section 4. The total surface area of the modeled source was consistent with the total surface area of contamination presented in Section 4. Thickness of contamination was based on sampling results. Retained site source areas were oriented such that their sides were parallel to east-west or north south trending lines.

5.6.1.2 Hydraulic Properties of Surface Soil. Surface soil hydraulic properties were obtained from Baca et al. 1992 and reported in the GWSCREEN user's manual (Rood 1994). Default Track 1 and 2 moisture contents for an annual infiltration rate of 10 cm/y were estimated from these data. Infiltration rates varied between source areas based on the most realistic future use of the retained sites. Therefore, the default moisture content could not be used. Surface soil hydraulic properties were estimated using the van Genuchten fitting parameters, α, and n, and other soil properties reported in Baca et al. 1992 (Table 5-4). These properties were assumed to represent the soil underlying the open ditches and surface contaminated areas. Sediments underlying the Industrial Waste Pond were modeled differently because the pond was designed to allow waste water to infiltrate. Moisture contents for these sediments were based on moisture characteristic curves presented in the Test Reactor Area RI/FS (EG&G Idaho 1991) for infiltration ponds present at that location. The pond is estimated to receive 2.2×10^5 m^3 of liquid effluent per year for the next 2 years and approximately 6,300 m^3 per year for subsequent years (see next section). A moisture content of 0.35 was assigned to the sediments for the first 2 years and 0.125 for all subsequent years.

5.6.1.3 Infiltration Rates for Ponds and Open Ditches. For ponds and open ditches that contain contaminated soil, their respective infiltration rates provide the driving force for the transport of contaminants from beneath these surface features, through the remaining vadose zone, and into the aquifer. These infiltration rates result from natural and anthropogenic sources. Natural sources of infiltrating water include precipitation as either rain that falls directly on to the ditch or pond surface or snowmelt from snow that falls directly on the surface feature. Natural infiltrating water can also include rainfall or snowmelt

from other areas of the site that may flow as runoff and collect in the ditches and reach the IWP. Indeed, several ditches at ANL-W that were never contaminated with effluent discharges relating to laboratory

Table 5-4. Hydraulic properties of surface soils (from Baca et al. 1992)

Parameter	Value
Saturated Hydraulic Conductivity (m/y)	23.9
Bulk density (g/cm^3)	1.5
Total Porosity (m^3/m^3)	0.487
Residual Moisture Content (m^3/m^3)	0.142
n	1.523
α (1/m)	1.066

operations, were constructed for the sole purpose of collecting surface runoff. Other ditches, once used for transfer of contaminated liquid effluent, now serve as runoff transfer ditches, such as the Interceptor Canal (ANL-09).

Anthropogenic sources of ditch or pond infiltration result from liquid effluent that results from normal operational processes at ANL-W. Historical flows in ANL-35 (North Ditch) averaged about 30 gpm. Currently, water still flows in the North Ditch as a part of ongoing effluent discharges. These current flows are at a rate of 5–15 gpm with an average of about 9 gpm (Martin 1996). The current flow rate of 5–15 gpm will be used in the modeling for future scenarios. Most of this water infiltrates the ditch before reaching the IWP.

The natural background infiltration rate for the ponds and ditches is a function of precipitation, evapotranspiration, and other factors. The widely accepted infiltration rate for the INEEL is taken from Track 2 guidance (DOE 1994) as 0.1 m/yr. This rate is assumed for all ditches and the IWP that contain known contamination whether they exist as open ditches or have been backfilled.

Runoff at ANL-W is reported to occur only upon certain conditions; i.e., when there is a rapid snowmelt in the spring but the top of the surface soil is still frozen, the frozen surface is impermeable and prevents infiltration of the snowmelt which then runs off (ANL-W 1996). However, the main parking lot at ANL-W is asphalt which also prevents infiltration of precipitation. The interceptor canal now serves to collect runoff from this parking lot. Other impermeable surfaces exist at ANL-W which can contribute precipitation as runoff. An assumption was made that the top surfaces of all buildings at the ANL-W have the potential to collect precipitation and that this precipitation is routed via gutters and downspouts to the open ditches that collect runoff.

Several steps were taken to determine the amount of precipitation that becomes runoff at ANL-W:

- Computed total surface area that potentially contributes runoff to ditches and canals
- Computed total area of impermeable surfaces within the runoff area of interest

- Assume impermeable surfaces have runoff equal to 100% of precipitation. Assumed permeable surfaces have only partial runoff of precipitation. The fraction of runoff was determined as the average (5.94%) of ten years of modeled runoff/precipitation fractions; these fractions were modeled using the Hydrologic Evaluation for Landfill Performance model (EPA 1994)

- Calculated annual runoff based on an average precipitation rate, total permeable surface area, total impermeable surface area, and the average runoff/precipitation ratio

- Distribute this total annual runoff among the ditches and canals on the basis of ditch or canal percentage of the combined ditch and canal length

- For each ditch, divided the portion of runoff received by the surface area of the ditch (assumed to be product of ditch length and width). This yields an individual ditch infiltration rate, assuming all runoff infiltrates within the ditch. This infiltration rate is in excess of the natural infiltration rate (0.1 m/y).

To determine the total surface area of interest receiving precipitation at ANL-W, the administrative boundary of ANL-W was arbitrarily chosen as the area potentially contributing runoff to the existing ditches and canal. The IWP was not included in the calculation of infiltration rates since it is the recipient of any runoff that does not fully infiltrate the ditch it flows in; however, it was assumed all runoff would infiltrate the ditch or canal it flows in before reaching the IWP. Still, the surface area of the IWP was not subtracted from the total surface area of interest since the IWP stage fluctuates thereby adding much uncertainty in the surface area representing the IWP. The total area within the administrative boundary of the ANL-W was determined to be 28,2950 m^2. The total impermeable surface area includes the main parking lot and the tops of major facilities and other paved or concrete surfaces.

Precipitation was assumed to be equal to the 35-yr average precipitation rate as measured by the National Oceanic and Atmospheric Administration using data collected at the INEEL Central Facilities Area weather station. The HELP model (Hydrologic Evaluation of Landfill Performance, EPA 1994) was used to calculate the annual runoff to precipitation ratio based on the given soil types and meteorological conditions (precipitation and temperature). This ratio was prepared for each of ten years as shown in Table 5-5. The ten-year average was used to determine the amount of precipitation that does not infiltrate and drains to one of the open ditches or canals.

The product of the ten year average runoff/precipitation ratio, the total permeable surface area, and the 35-year average precipitation rate yields the amount of runoff contributed from permeable surfaces at ANL-W to the open ditches and canals. The total area represented by currently open ditches and canal was subtracted from the total permeable area. However, infiltration from precipitation that falls directly on the open ditches and canals was added into the total infiltration rate. The product of the 35-year average annual precipitation rate and the total impermeable surface area yields the total annual runoff from impermeable surfaces. Table 5-6 presents a summary of the permeable and impermeable surface areas.

Table 5-5. Runoff/precipitation ratios based on HELP model output.

Year	Runoff/precipitation
1983	3.78E–02
1984	7.27E–02
1985	1.23E–01
1986	9.58E–02
1987	8.03E–02
1988	3.00E–02
1989	5.65E–02
1990	4.81E–02
1991	4.05E–02
1992	8.98E–03
10-year average	5.94%

Table 5-6. Calculation of areas involved in runoff at ANL-W.

Subunit	Area (m^2)	Annual runoff[a] (m^3)
Potential runoff area	282,950	
Impermeable area (excluding parking lot)	34,819	7,660
Parking lot[b]	9,275	2,041
Total impermeable area	44,094	9,701
Permeable area (including ditches and canals)	238,856	
Active ditch and canal area	3,679	
Total permeable area	235,177	3,073
Total runoff (excluding parking area)		10,733

(a) Based on the product of surface area times 0.22 m annual precipitation. For permeable surfaces, runoff is multiplied by the average runoff to precipitation ratio of 0.0594

(b) Parking lot runoff only added to ANL-09 infiltration

Finally, a method was implemented for the distribution of the total annual runoff among the open ditches at ANL-W that are designed or currently used for collection of such runoff. A simple scheme was employed based on the surface area of each ditch and canal. All of the main parking lot runoff was routed to the interceptor canal (ANL-09) because this canal was the most likely recipient. The remaining runoff

was then divided among the canal and ditches based on their surface area relative to the total surface area of all ditches and canals. An additional conservative assumption was made to ignore the several ditches that are not contaminated with any known ANL-W related contamination but that were put in place to collect runoff. Therefore, all remaining runoff is distributed among the canal and ditches known to contain site-related contamination. The infiltration from runoff was added to the natural infiltration from precipitation that falls directly on the ditches and canals (0.1 m/y). Table 5-7 summarizes the runoff distribution and the determination of final infiltration rates for the open ditches and canal at ANL-W. Since it is known that ANL-01 Ditch B has been backfilled during past remedial efforts, this ditch was omitted from the combined site canal and ditch length and also from the distribution of runoff.

Table 5-7. Runoff distribution and final infiltration rates for open ditches and canal.

Unit	Length (m)	Width (m)	Area (m^2)	Percent of Total Area	Annual water flux via runoff (m^3/yr)	Annual water flux via precipitation (m^3/yr)	Total water flux to ditch or canal (m^3/yr)	Annual infiltration (m/yr)
ANL-01 ditch A element 1	91	1.5	136.5	3.7	398	13.7	412	3.02
element 2	41	1.5	61.5	1.7	179	6.15	185	3.02
ANL-01 ditch C	1.5	152	228	6.2	665	22.8	688	3.02
ANL-01A element 1	1.8	158	284	7.7	829	28.4	858	3.02
element 2	1.8	31	55.8	1.5	162	5.58	168	3.02
ANL-09	9.1	300	2,730	72.4	9,479[a]	273	9752	3.50
ANL-35	1.2	152	184	5	532	18.2	24,444[b]	134

(a) Annual runoff water flux includes all runoff from parking area (2,041 $m^3\ y^{-1}$)

(b) Total water flux for ANL-35 includes an effluent flow rate for routine operations of 10 gal min^{-1} (23,893 $m^3\ y^{-1}$). This flow rate was assumed to be the same for 100 years.

5.6.2 Unsaturated Zone Parameters

5.6.2.1 Unsaturated Thickness. Interbed thickness is known to vary across ANL-W, and well logs have been used to delineate thickness at different locations. Isopleths of interbed thickness are plotted in Figure 5-3. Isopleths were generated using a Kriging interpolation routine that is part of the Surfer® software package (Golden Software Inc. 1996). All source areas are underlain by sedimentary interbeds of varying thickness. An interbed thickness (Table 5-8) was assigned to each source area based on the isopleths plotted in Figure 5-3.

5.6.2.2 Hydraulic Properties of Interbeds. Hydraulic properties of the interbeds underlying ANL-W were not available. Therefore, hydraulic properties were obtained from the modeling effort for the Radioactive Waste Management Complex (Magnuson and Sondrup 1996). Van Genuchten fitting parameters and other hydraulic properties for the B-C interbed were assumed to represent hydraulic properties of sedimentary interbeds beneath ANL-W (Table 5-9).

Table 5-8. Interbed thickness for wells in the vicinity of ANL-W and interpolated interbed thickness for each source area.

Well Number	UTM East (m)	UTM North (m)	Total Unsaturated Interbed Thickness (m)	Source Area	Total Unsaturated Interbed Thickness (m)
Arbor Test Well	366,998	4,827,180	48.2	ANL 09 Ditch	15
EBR-II #1	366,338	4,828,287	25.3	ANL-09 Mound	15
EBR-II #2	366,180	4,828,293	22.6	ANL-01 B Ditch Element 1	15
MW-11	366,057	4,828,003	7.6	ANL-01 B Ditch Element 2	18
MW-12	367,068	4,829,182	49.1	ANL-01 C Ditch	16
MW-13	365,824	4,828,338	8.7	ANL-08 EBR Leach Pit	18
SITE 16	366,404	4,828,295	56.4	ANL-01A Element 1	17
USGS 100	365,229	4,827,024	32.3	ANL-01A Element 2	17
MW-14	365,889	4,828,072	6.7	ANL-01 Ditch A Element 1	20
				ANL-01 Ditch A Element 2	22
				ANL-35 North Ditch	25
				ANL-53 Riser Pits	20
				ANL-01 Industrial Waste Pond	17

Table 5-9. Hydraulic properties of interbeds (from Magnuson Sondrup, 1996)

Parameter	Value
Saturated Hydraulic Conductivity (m/y)	1.26
Total Porosity (m^3/m^3)	0.48
Residual Moisture Content (m^3/m^3)	0.083
Bulk density (g/cm^3)	1.5
n	2.534
α (1/m)	3.196

5.6.3 Saturated Zone Parameters

Saturated zone parameters were obtained from the Track 1 and 2 guidance manuals (DOE 1992 and 1994) and site-specific data (Table 5-10). Values for effective porosity, dispersivity, and well screen thickness were obtained from the TRACK 1 and 2 guidance manuals. A pore velocity (average linear velocity) for the Snake River Plain Aquifer near ANL-W of 360 m/y was obtained from LMITCO (1995). The effective aquifer thickness was assigned a value of 76 m based on the work of Arnett et al. (1990) and presented in Maheras et al. (1994).

The aquifer model used in this analysis included dispersion in three directions. Therefore, a vertical dispersivity value needed to be assigned. Typically, the vertical dispersivity is assumed to be the same as the transverse dispersivity. In order to provide some conservatism to the calculation, vertical dispersivity was assigned a value 10 times less than the transverse dispersivity (0.4 m). Contaminants were allowed to disperse into the effective thickness of the aquifer (76 m). Output concentrations were based on the average aquifer concentration in the first 15-m of aquifer (the well screen thickness) measured from the surface. Near the source, little vertical mixing occurs and calculated concentrations are similar to those calculated using a vertically averaged model and 15-m mixing thickness. Note, this is the same approach used in the Track 1 and 2 process. Farther away from the source, the contaminant plume disperses beyond the 15-m well screen depth. Therefore, contaminant concentrations are lower compared to those calculated assuming a constant, 15-m mixing thickness.

Maximum concentrations in the aquifer were calculated for a receptor located on the downgradient edge of each source. Concentrations were also calculated at 100 years for several key contaminants (arsenic, chromium). One-hundred year contaminant concentrations were calculated at each node on the receptor grid (814 nodes). Contaminant concentrations at each receptor node were calculated separately for each source and then summed across all sources. Therefore, concentrations for a specific contaminant at each receptor node represents contributions from all sources considered in the assessment.

Table 5-10. Transport parameter values for the saturated zone model.

Parameter	Value
Pore (average linear) velocity (m/y)	360
Effective porosity (m^3/m^3)	0.1
Bulk Density (g/cm^3)	1.9
Effective aquifer thickness (m)	76
Longitudinal dispersivity (m)	9
Transverse dispersivity (m)	4
Vertical dispersivity (m)	0.4
Well screen thickness (m)	15

5.7 Contaminant Inventory and Screening

5.7.1 Screening Procedures

Prior to modeling the groundwater exposure pathway, soil contamination data were screened to eliminate low-risk contaminants and minimize the modeling input. Initially, statistical analyses were prepared on the large set of soil sampling results to obtain such statistics as mean concentration, standard deviation, and 95% upper confidence limit (UCL) for each detected contaminant at each soil contamination site (Appendix A). These statistics were used to eliminate those contaminants that are below accepted background soil concentration levels.

Those soil contaminants that exceed background levels were put through an additional screening process to minimize the modeling input for the groundwater pathway analysis. This secondary screening process compares the concentration of contaminated water within soil matrix pore spaces to screening level risk-based concentrations for drinking water. Pore water toxicity screening assumes (a) pores of soil are fully saturated with water, (b) soil porosity is uniformly distributed throughout the matrix, (c) all contamination is partitioned between the soil particle surfaces and water occupying the soil pore spaces, and (d) the receptor consumes water that is at the pore water concentration (i.e., the water is drawn from a well that is completed within the contaminated soil matrix).

Screening-level risk-based concentrations were obtained for ingestion of tap water from the EPA (EPA 1995). These reflect 10^{-6} risk-based concentrations (for carcinogens) or HQ = 0.1 based concentrations (for noncarcinogens). When no EPA-prepared risk-based concentrations or toxicity data were available to prepare risk-based concentrations, tap water maximum contaminant levels (MCLs), obtained from "Drinking Water Regulations and Health Advisories" (EPA 1995c), were used for comparison.

Pore-water screening assumes the contaminant present in the soil partitions between the solid soil particles and the liquid water phase occupying the pore spaces of the soil matrix. The amount of pre water present is assumed as a saturation level of 0.3 and the partitioning is based on the contaminant's retardation factor [given as $1+ Kd(\rho_b/\theta)$, a unitless value where Kd is the contaminant sorption coefficient, ρ_b is the soil matrix bulk density, θ is the water-filled porosity of the soil]. The contaminant concentration in the soil matrix expressed as mg of contaminant per kg of soil is converted to pore-water concentration using the following procedure; a) multiply the soil concentration by the soil bulk density (1.5 g/cm^3) and the appropriate conversion factors (1kg/10^{3}g and 1L/10^{3}cm^3) to obtain mg of contaminant per L of soil: b) next, the volumetric soil concentration is converted to volumetric pore-water concentration by assuming a saturation level of 0.3 (i.e., divide the volumetric soil concentration by the saturation level 0.3 to obtain pore-water volumetric concentration in mg of contaminant per L of pore-water): c) finally, the contaminant is allowed to partition between the solid and liquid phases (soil and pore-water) by dividing the pore-water volumetric concentration by the contaminant retardation factor, Rd, to obtain a partitioned pore-water volumetric concentration in mg of contaminant per L of pore-water. This value is then used for comparison against RBCs and MCLs. The final formula for this conversion is as follows:

$$C_{pw} = C_s*(\rho_b)/(\theta*R_d)*(1/1000)*(1000/1) \qquad (5\text{-}16)$$

where

C_{pw} = contaminant concentration in pore-water (mg/L)

C_s = contaminant concentration in soil (mg/kg)

ρ_b = soil bulk density (1.5 g/cm^3)

θ = water-filled porosity (saturation level) (0.3)

R_d = contaminant retardation factor ($1+Kd*\rho_b/\theta$)

and 1/1000 = conversion factor from grams to kilograms

1000/1 = conversion factor from cm^3 to L

An example of using equation 5-16 for the calculation of pore-water concentration, using the ANL-01—Industrial Waste Pond contaminant arsenic (soil concentration of 25 mg/kg, Kd = 3 mL/g), is as follows:

C_{pw} = 25 mg/kg*(1.5 g/cm^3)/{0.30*[1+(3 mL/g*1.5g/cm^3/0.3)]}*(1kg/1000g)*(1000cm^3/1L) = 7.81 mg/L

For radiological contaminants, with soil concentrations expressed in ρCi/g, the calculation is similar except for the conversion factors since these concentrations are usually expressed in ρCi/L. An example calculation of pore-water concentrations at ANL-01—Industrial Waste Pond using the contaminant Sr-90 (soil concentration = 2.5 ρCi/g, Kd = 24 mL/g) is as follows:

C_{pw} = 2.5ρCi/g *(1.5 g/cm^3)/{0.30*[1+(24 mL/g*1.5g/cm^3/0.3)]}*(1000cm^3/1L) = 1.03E+02 ρCi/L

The resulting partitioned pore water concentrations were then compared to limiting water concentration standards (i.e., the risk-based or hazard-based concentration, or MCL, depending on data availability). Those contaminants with a pore water concentration less than their corresponding limiting water concentration were eliminated from further consideration in the groundwater pathway modeling effort.

Initially, sampling results indicated a total of 55 unique contaminants in the contaminated soil sites at ANL-W, exclusive of the EBR-II Leach Pit. The analysis of EBR-II Leach Pit contaminant inventories is presented in a following section. The contaminated soil sites analyzed in this section include ANL-01 Industrial Waste Pond, Ditches A, B, and C; ANL-01A (Main Cooling Tower Blowdown Ditch); ANL-09 Interceptor Canal and soil mound; ANL-35 (North Ditch); ANL-53 Riser Pits, North Drainage Area, South Drainage Area; and ANL-61A (PCB-contaminated soil site). Many of the 55 contaminants are common to the various soil contamination sites (e.g., a total of 168 site-specific potential contaminants of concern were examined for the 12 sites listed above).

Screening the site-specific contaminant concentrations against INEEL background 95/95 UCL values eliminated 25 (15% of total) of these site-specific potential contaminants of concern. Screening of pore water concentrations against limiting water concentrations eliminated an additional 51 of the site-specific contaminants (31% of total). The pore water screening effort is summarized in Tables 5-11 through 5-22. The soil concentrations listed in these tables are the smallest concentration of either the UCL or the maximum concentration value from the sampling data set. The pore water screening process left a total of 91 site-specific contaminants of concern to be modeled. These 91 include 36 unique compounds, seven of which are retained due to a lack of data regarding their toxic effects (i.e., HpCDD, HpCDF,

HxCDF, O-Phosphate, OCDF, PeCDD, TCDF). Of the six dioxin furan compounds, the equivalent risk factors based on OCDD were used to calculate the actual risk of the groundwater in the human health risk assessment (Appendix B).

5.7.2 Calculation of Contaminant Inventories in Source Areas

The pore water screening process left a total of 36 unique contaminants of concern (91 site-specific contaminants of concern) to be modeled. These 91 include one contaminant (O-Phosphate) retained due to a lack of data regarding its toxic effects. To determine the potential contaminant inventory at each of the 12 soil sites, the total contaminated soil per each site was estimated. This estimate is simply the product of the site length, width, and thickness of contaminated soil. Although these dimensions certainly vary (i.e., the sites are not perfect rectangles), they are here assumed constant. In particular, different contaminants are known to extend vertically into the subsurface to different depths due to differences in abundance or chemical properties. However, the extent of contamination is assumed to be constant for the suite of contaminants at a particular subsite. Additionally, it is assumed that each contaminant is uniformly distributed throughout the contaminated volume of soil. Other properties, such as soil bulk density (1.5 g/cm^3), are assumed uniformly distributed in determining the contaminated soil volume and mass. The dimensions for the sites and their associated contaminated soil volumes and masses are presented in Table 5-23.

Final contaminant of concern inventories at each subsite were calculated as the product of the unscreened contaminant concentrations presented in Tables 5-11 through 5-22 and the corresponding contaminated soil mass given in Table 5-23. The resulting inventories are presented for each contaminant of concern at each site in Table 5-24.

Several sites were represented as two area source elements (ANL-01 ditch A, ANL-01 ditch B, and ANL-01A) Inventories in each source element were distributed based on the relative surface area of the element.

5.7.3 EBR-II Leach Pit Source Term

The ANL-08 (EBR-II Leach Pit) required additional modeling to estimate release to the subsurface. The leach pit was operated from 1958 to 1973 (15 years). After 1973, the pit received a small one-time release of tritiated water. The EBR-II Leach Pit was blasted out of basalt and a concrete wall and a concrete cap remained over the top of the pit. In 1991, sludge from the pit was sampled, and 7.34 m^3 of sludge was removed in 1993. A small amount of sludge (less than 1/16-th of an inch) remained in the bottom of the pit. After sludge removal, the pit was capped with bentonite. Our problem was to assess migration of contaminants from the pit while the pit was operating (i.e., the only driving force of contaminants).

Past disposal records were inaccurate and inconsistent with the measured concentrations of contaminants in the sludge removed. For example, several nuclides were reported to have more activity removed from the pit than what was disposed according to the disposal records. For these reasons, the past disposal records were not used to reconstruct past releases. However, the estimated volumes of liquid effluent reported in the disposal records were used in the reconstruction calculations. The conceptual model illustrated in Figure 5-4 was used to reconstruct past releases from the pit.

Table 5-11. Screening of ANL-01—Industrial Waste Pond soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Arsenic	25	2.65E+07	3	7.81E+00	4.7E-05	No
Cadmium	3.76	3.99E+06	6	6.06E-01	1.8E-02	No
Chloride	41.44	4.40E+07	0	2.07E+02	ND	Yes
Chromium	11,400	1.21E+10	1.2	8.14E+03	1.8E-01	No
Copper	76.13	8.08E+07	20	3.77E+00	1.4E+00	No
Fluoride	9.05	9.61E+06	0	4.53E+01	2.2E+00	No
Mercury	0.86	9.13E+05	100	8.58E-03	1.1E-02	Yes
Nitrate	3.1	3.29E+06	0.007	1.50E+01	5.8E+01	Yes
O-Phosphate	1.3	1.38E+06	ND	6.50E+00	ND	No
Selenium	8.41	8.93E+06	4	2.00E+00	1.8E-01	No
Silver	31.79	3.38E+07	90	3.52E-01	1.8E-01	No
Sulfate	3,300	3.50E+09	0	1.65E+04	5.0E+02	No
Zinc	1,633.3	1.73E+09	16	1.01E+02	1.1E+01	No
Cm-244	0.11	1.17E+08	6	1.77E+01	2.3E-01	No
Co-60	0.117	1.24E+08	10	1.15E+01	2.5E+00	No
Cs-137	29.2	3.10E+10	500	5.84E+01	1.5E+00	No
Sr-90	2.5	2.65E+09	24	1.03E+02	8.5E-01	No
1,1,1-Trichlorethane	0.0178	1.89E+04	0.312	3.48E-02	3.3E+00	Yes
2, 4,5-Tp (Silvex)	27.6	2.93E+07	ND	1.38E+02	4.0E+00	No
2-Butanone	0.2	2.12E+05	0.013	9.37E-01	2.2E+01	Yes
Acetone	0.13	1.38E+05	0.001	6.46E-01	3.7E+00	Yes
Chloroform	0.0077	8.18E+03	0.131	2.33E-02	1.4E-02	No
Di-n-butylphthalate	0.41	4.35E+05	4.14	9.45E-02	3.7E+00	Yes
Diethylphthalate	0.41	4.35E+05	0.208	1.00E+00	2.9E+01	Yes
Methylene Chloride	0.295	3.13E+05	0.026	1.30E+00	1.1E-02	No
Toluene	0.0016	1.70E+03	0.9	1.45E-03	7.3E+00	Yes
bis (2-Ethylhexyl) phthalate	0.008	8.50E+03	18	4.40E-04	6.1E-03	Yes

Table 5-12. Screening of ANL-01—Ditch A soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Arsenic	11.7	1.89E+06	3	3.66E+00	4.7E-05	No
Chromium	79.41	1.28E+07	1.2	5.67E+01	1.8E-01	No
Copper	34.74	5.61E+06	20	1.72E+00	1.4E+00	No
Cyanide	0.45	7.26E+04	0.00173	2.23E+00	7.3E-01	No
Mercury	0.719	1.16E+05	100	7.18E-03	1.1E-02	Yes
Silver	0.862	1.39E+05	90	9.56E-03	1.8E-01	Yes
Thallium	0.792	1.28E+05	0	3.96E+00	3.0E-03	No
Zinc	243.35	3.93E+07	16	1.50E+01	1.1E+01	No
Sr-90	4.5	7.26E+08	24	1.86E+02	8.5E-01	No
U-238	5.8	9.36E+08	6	9.35E+02	7.7E-01	No

Table 5-13. Screening of ANL-01—Ditch B soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Arsenic	7.51	2.90E+06	3	2.35E+00	4.7E-05	No
Chromium	1,183.82	4.58E+08	1.2	8.46E+02	1.8E-01	No
Copper	125.87	4.87E+07	20	6.23E+00	1.4E+00	No
Lead	23.31	9.01E+06	100	2.33E-01	1.5E-02	No
Mercury	0.838	3.24E+05	100	8.36E-03	1.1E-02	Yes
Selenium	2.168	8.38E+05	4	5.16E-01	1.8E-01	No
Silver	0.747	2.89E+05	90	8.28E-03	1.8E-01	Yes
Zinc	3,020	1.17E+09	16	1.86E+02	1.1E+01	No
1,1,1-Trichlorethane	0.0043	1.66E+03	0.312	8.40E-03	3.3E+00	Yes
2, 4-D	0.087	3.36E+04	ND	4.35E-01	7.3E-02	No
Acetone	0.025	9.66E+03	0.0011	1.24E-01	3.7E+00	Yes
Di-n-butylphthalate	0.35	1.35E+05	4.14	8.06E-02	3.7E+00	Yes
Methylene Chloride	0.021	8.12E+03	0.0261	9.29E-02	1.1E-02	No
bis (2-Ethylhexyl) phthalate	0.12	4.64E+04	18	6.59E-03	6.1E-03	No

ND indicates no data available.

Table 5-14. Screening of ANL-01—Ditch C soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Arsenic	7.62	2.02E+06	3	2.38E+00	4.7E–05	No
Chloride	37	9.82E+06	ND	1.85E+02	ND	Yes
Cyanide	5.44	1.44E+06	0.00173	2.70E+01	7.3E–01	No
Fluoride	7	1.86E+06	0	3.50E+01	2.2E+00	No
Mercury	0.177	4.70E+04	100	1.77E–03	1.1E–02	Yes
Nitrate	9.5	2.52E+06	0.007	4.59E+01	5.8E+01	Yes
O-Phosphate	17	4.51E+06	ND	8.50E+01	ND	No
Selenium	1.706	4.53E+05	4	4.06E–01	1.8E–01	No
Silver	1.22	3.24E+05	90	1.35E–02	1.8E–01	Yes
Sulfate	620	1.65E+08	0	3.10E+03	5.0E+02	No
Thallium	0.89	2.36E+05	0	4.45E+00	3.0E–03	No
U-238	21	5.58E+09	6	3.39E+03	7.7E–01	No
1,1,1-Trichlorethane	0.056	1.49E+04	0.312	1.09E–01	3.3E+00	Yes
2-Butanone	0.11	2.92E+04	0.0135	5.15E–01	2.2E+01	Yes
Acetone	0.069	1.83E+04	0.00111	3.43E–01	3.7E+00	Yes
Chloroform	0.01	2.65E+03	0.131	3.02E–02	1.4E–02	No
Methylene Chloride	0.17	4.51E+04	0.0261	7.52E–01	1.1E–02	No
Toluene	0.0079	2.10E+03	0.9	7.18E–03	7.3E+00	Yes

ND indicates either no sorption coefficient data or no toxicity data available.

Table 5-15. Screening of ANL-01A—MCTBD soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Antimony	18.62	6.64E+06	50	3.71E-01	1.5E-02	No
Arsenic	16.55	5.91E+06	3	5.17E+00	4.7E-05	No
Chromium	204.94	7.31E+07	1.2	1.46E+02	1.8E-01	No
Copper	92.36	3.30E+07	20	4.57E+00	1.4E+00	No
Cyanide	0.81	2.89E+05	0.00173	4.02E+00	7.3E-01	No
Lead	35.69	1.27E+07	100	3.56E-01	1.5E-02	No
Mercury	1.57	5.60E+05	100	1.57E-02	1.1E-02	No
Selenium	0.82	2.93E+05	4	1.95E-01	1.8E-01	No
Silver	6.43	2.29E+06	90	7.13E-02	1.8E-01	Yes
Zinc	279.13	9.96E+07	16	1.72E+01	1.1E+01	No
U-238	2.7	9.63E+08	6	4.35E+02	7.7E-01	No
2, 4-D	0.0064	2.28E+03	ND	3.20E-02	7.3E-02	Yes
Acetone	0.046	1.64E+04	0.00111	2.29E-01	3.7E+00	Yes
Di-n-butylphthalate	0.12	4.28E+04	4.14	2.76E-02	3.7E+00	Yes
Di-n-octylphthalate	0.048	1.71E+04	2,930,000	1.64E-08	7.3E-01	Yes
Diethylphthalate	0.002	7.14E+02	0.208	4.90E-03	2.9E+01	Yes
Methylene Chloride	0.037	1.32E+04	0.0261	1.64E-01	1.1E-02	No
bis (2-Ethylhexyl) phalate	0.096	3.43E+04	18	5.27E-03	6.1E-03	Yes

ND indicates no sorption coefficient data available.

Table 5-16. Screening of ANL-09—Canal soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Arsenic	9.65	1.05E+08	3	3.02E+00	4.7E-05	No
Mercury	0.173	1.88E+06	100	1.73E-03	1.1E-02	Yes
Silver	1.47	1.60E+07	90	1.63E-02	1.8E-01	Yes
Co-60	0.039	4.25E+08	10	3.82E+00	2.5E+00	No
Cs-134	0.021	2.29E+08	500	4.20E-02	1.0E+00	Yes
Cs-137	18	1.96E+11	500	3.60E+01	1.5E+00	No

Table 5-17. Screening of ANL-09—Mound contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Am-241	0.034	5.78E+07	340	9.99E-02	1.4E-01	Yes
Cm-244	0.06	1.02E+08	6	9.68E+00	2.3E-01	No
Co-60	0.198	3.36E+08	10	1.94E+01	2.5E+00	No
Cs-137	30.53	5.19E+10	500	6.10E+01	1.5E+00	No
U-238	2.3	3.91E+09	6	3.71E+02	7.7E-01	No

Table 5-18. Screening of ANL-35—North Ditch soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Chromium	55.45	4.71E+06	1.2	3.96E+01	1.8E-01	No
Cobalt	8.83	7.50E+05	10	8.66E-01	2.2E+00	Yes
Copper	116.29	9.88E+06	20	5.76E+00	1.4E+00	No
Cyanide	3.93	3.34E+05	0.00173	1.95E+01	7.3E-01	No
Fluoride	4.8	4.08E+05	0	2.40E+01	2.2E+00	No
Mercury	0.24	2.04E+04	100	2.40E-03	1.1E-02	Yes
Nitrate	22	1.87E+06	0.007	1.06E+02	5.8E+01	No
O-Phosphate	2.5	2.12E+05	0	1.25E+01	ND	No
Selenium	0.73	6.20E+04	4	1.74E-01	1.8E-01	Yes
Silver	0.73	6.20E+04	90	8.09E-03	1.8E-01	Yes
Strontium	63	5.35E+06	24	2.60E+00	2.2E+01	Yes
Sulfate	140	1.19E+07	0	7.00E+02	5.0E+02	No
Zinc	230.26	1.96E+07	16	1.42E+01	1.1E+01	No
Co-60	0.037	3.14E+06	10	3.63E+00	2.5E+00	No
Cs-137	2	1.70E+08	500	4.00E+00	1.5E+00	No
H-3	0.719	6.11E+07	0	3.60E+03	6.6E+02	No
1,1,1-Trichlorethane	0.53	4.50E+04	0.312	1.04E+00	3.3E+00	Yes
2, 4-D	0.091	7.73E+03	ND	4.55E-01	7.3E-02	No
2-Butanone	0.18	1.53E+04	0.0135	8.43E-01	2.2E+01	Yes
Acetone	0.22	1.87E+04	0.00111	1.09E+00	3.7E+00	Yes
Acetonitrile	0.0776	6.59E+03	ND	3.88E-01	2.2E-01	No
Butylbenzyl-phthalate	0.074	6.29E+03	0.203	1.84E-01	7.3E+00	Yes
Chloroform	0.0061	5.18E+02	0.131	1.84E-02	1.4E-02	No
Di-n-butylphthalate	0.23	1.95E+04	4.14	5.30E-02	3.7E+00	Yes
Di-n-octylphthalate	0.49	4.16E+04	2930000	1.67E-07	7.3E-01	Yes
HpCDD	4.04E-06	3.43E-01	ND	2.02E-05	ND[1]	No
HpCDF	4.7E-07	3.99E-02	ND	2.35E-06	ND[1]	Yes
HxCDF	2.9E-07	2.46E-02	ND	1.45E-06	ND[1]	No
OCDD	9.99E-06	8.49E-01	ND	5.00E-05	ND[1]	No
OCDF	5.8E-07	4.93E-02	ND	2.90E-06	ND[1]	No
PeCDD	2.2E-07	1.87E-02	ND	1.10E-06	ND[1]	No

Table 18. (continued).

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
TCDF	4.7E-07	3.99E-02	ND	2.35E-06	ND[1]	No
Methylene Chloride	0.21	1.78E+04	0.0261	9.29E-01	1.1E-02	No
Toluene	0.01	8.50E+02	0.9	9.09E-03	7.3E+00	Yes
bis (2-Ethylhexyl) phthalate	0.37	3.14E+04	18	2.03E-02	6.1E-03	No

ND indicates no sorption coefficient or no toxicity data available.

1 indicates that RBC will be calculated on OCDD value equivalence.

Table 5-19. Screening of ANL-53—Riser Pits soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Arsenic	76	8.07E+04	3	2.38E+01	4.7E-05	No
Chromium	1717	1.82E+06	1.2	1.23E+03	1.8E-01	No
Lead	4725	5.02E+06	100	4.72E+01	1.5E-02	No
Mercury	0.78	8.28E+02	100	7.78E-03	1.1E-02	Yes

Table 5-20. Screening of ANL-53—North Drainage area soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Chromium	59	1.88E+05	1.2	4.21E+01	1.8E-01	No
Lead	43.1	1.37E+05	100	4.30E-01	1.5E-02	No
Mercury	0.4	1.27E+03	100	3.99E-03	1.1E-02	Yes

Table 5-21. Screening of ANL-53—South Drainage area soil contamination for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore Water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
Chromium	56	1.78E+05	1.2	4.00E+01	1.8E-01	No
Mercury	0.086	2.74E+02	100	8.58E-04	1.1E-02	Yes

Table 5-22. Screening of ANL-61A soil contamination site for groundwater pathway modeling.

Compound	Soil Concentration (mg/kg or pCi/g)	Total Mass (mg or pCi)	Kd (mL/g)	Pore water Concentration (mg/L or pCi/L)	RBC (mg/L or pCi/L)	Screen (Yes or No)
PCBs	55	3.68E+06	100	5.49E-01	8.7E-06	No

Table 5-23. Soil contamination sites and dimensional data.

	Name	Length (m)	Width (m)	Thickness (m)	Volume (m^3)	Contaminated Mass (kg)[a]
ANL-09[b]	Interceptor canal	434.3	9.1	1.8	7114	1.07E+07
	Mounded soil	152.4	6.1	1.2	1116	1.67E+06
ANL-35	North ditch	152.4	1.2	0.3	55	8.23E+04
ANL-01	Ditch A[c]	121.9	1.5	0.6	104	1.56E+05
	Ditch B[c]	426.7	1.5	0.4	250	3.74E+05
	Ditch C	152.4	1.5	0.76	174	2.61E+05
	Industrial Waste Pond	76.2	61.0	0.15	696	1.04E+06
ANL-01A[b]	Main Cooling Tower Blowdown Ditch	213.4	1.8	0.6	233	3.49E+05
ANL-53	Cooling tower riser pits	0.4	4.9	0.4	0.7	1.05E+03
	North drain area	3.0	1.8	0.4	2.1	3.08E+03
	South drain area	3.0	1.8	0.4	2.1	3.08E+03
ANL-61A	PCB-contaminated area	6.4	4.6	1.5	44	6.58E+04

a. Contaminated mass represents upper bound of potentially contaminated soil based on bulk density of 1.5 g/cm^3.
b. Only the active area (first 300 m) of ANL-09 ditch was modeled.
c. These units were modeled as two separate areas.

Table 5-24. List of final contaminants of concern and corresponding masses (mg or pCi) for groundwater pathway modeling.

Compound	ANL-01 Industrial Waste Pond	ANL-01 Ditch A[a]	ANL-01 Ditch B[b]	ANL-01 Ditch C	ANL-01A[c] MCTBD[d]	ANL-09 Interceptor canal	ANL-09 Mounded soil	ANL-35 North ditch	ANL-53 Riser Pits	ANL-53 North drain area	ANL-53 South drain area	ANL-61A PCB site
Inorganics												
Antimony	—[1]	—	—	—	6.64E+06	—	—	—	—	—	—	—
Arsenic	2.65E+07	1.89E+06	2.90E+06	2.02E+06	5.91E+06	1.05E+08	—	—	8.07E+04	—	—	—
Cadmium	3.99E+06	—	—	—	—	—	—	—	—	—	—	—
Copper	8.08E+07	5.61E+06	4.87E+07	—	3.30E+07	—	—	9.88E+06	—	—	—	—
Chromium	1.21E+10	1.28E+07	4.58E+08	—	7.31E+07	—	—	4.71E+06	1.82E+06	1.88E+05	1.78E+05	—
Cyanide		7.26E+04	—	1.44E+06	2.89E+05	—	—	3.34E+05	—	—	—	—
Fluoride	9.61E+06	—	—	1.86E+06	—	—	—	4.08E+05	—	—	—	—
Lead	—	—	9.01E+06	—	1.27E+07	—	—	—	5.02E+06	1.37E+05	—	—
Mercury	—	—	—	—	5.60E+05	—	—	—	—	—	—	
Nitrate	—	—	—	—	—	—	—	1.87E+06	—	—	—	—
Selenium	8.93E+06	—	8.38E+05	4.53E+05	2.93E+05	—	—	—	—	—	—	—
Silver	3.38E+07	—	—	—	—	—	—	—	—	—	—	—
Sulfate	3.50E+09	—	—	1.65E+08	—	—	—	1.19E+07	—	—	—	—
Thallium	—	1.28E+05	—	2.36E+05	—	—	—		—	—	—	—
Zinc	1.73E+09	3.93E+07	1.17E+09	—	9.96E+07	—	—	1.96E+07	—	—	—	—
Organics												
Acetonitrile	—	—		—	—	—	—	6.59E+03	—	—	—	—
bis (2-Ethylhexyl) phalate	—	—	4.64E+04	—	—	—	—	3.14E+04	—	—	—	—
Chloroform	8.18E+03	—	—	2.65E+03	—	—	—	5.18E+02	—	—	—	—
Methylene Chloride	3.13E+05	—	8.12E+03	4.51E+04	1.32E+04	—	—	1.78E+04	—	—	—	—
PCBs	—	—	—	—	—	—	—	—	—	—	—	3.68E+06
HpCDD	—	—	—	—	—	—	—	3.43E-01	—	—	—	—

Table 5-24. (continued).

Compound	ANL-01 Industrial Waste Pond	ANL-01 Ditch A[a]	ANL-01 Ditch B[b]	ANL-01 Ditch C	ANL-01A[c] MCTBD[d]	ANL-09 Interceptor canal	ANL-09 Mounded soil	ANL-35 North ditch	ANL-53 Riser Pits	ANL-53 North drain area	ANL-53 South drain area	ANL-61A PCB site
HpCDF	---	---	---	---	---	---	---	3.99E-02	---	---	---	---
HxCDF	---	---	---	---	---	---	---	2.46E-02	---	---	---	---
O-Phosphate	1.38E+06	---	---	4.51E+06	---	---	---	2.12E+05	---	---	---	---
OCDD	---	---	---	---	---	---	---	8.49E-01	---	---	---	---
OCDF	---	---	---	---	---	---	---	4.93E-02	---	---	---	---
PeCDD	---	---	---	---	---	---	---	1.87E-02	---	---	---	---
TCDF	---	---	---	---	---	---	---	3.99E-02	---	---	---	---
2, 4,5-Tp (Silvex)	2.93E+07	---	---	---	---	---	---	---	---	---	---	---
2, 4-D	---	---	3.36E+04	---	---	---	---	7.73E+03	---	---	---	---
Radionuclides												
Cm-244	1.17E+08	---	---	---	---	---	1.02E+08	---	---	---	---	---
Co-60	1.24E+08	---	---	---	---	4.25E+08	3.36E+08	3.14E+06	---	---	---	---
Cs-137	3.10E+10	---	---	---	---	1.96E+11	5.19E+10	1.70E+08	---	---	---	---
H-3	---	---	---	---	---	---	---	6.11E+07	---	---	---	---
Sr-90	2.65E+09	7.26E+08	---	---	---	---	---	---	---	---	---	---
U-238	---	9.36E+08	---	5.58E+09	9.63E+08	---	3.91E+09	---	---	---	---	---

1. --- indicates the compound does not exist at this site above risk-based concentration level.
a. Represented as 2 area source elements. The element elongated north-south contained 69% of the inventory. The remainder was apportioned to the east-west elongated element.
b. Represented as 2 area source elements. The element elongated north-south contained 69% of the inventory. The remainder was apportioned to the east-west elongated element.
c. Represented as 2 area source elements. The larger element contained 84% of the inventory. The remainder was apportioned to the smaller element.
d. MCTBD (Main Cooling Tower Blowdown Ditch).

The change in contaminant inventory in the pit with respect to time is described by the differential equation

$$\frac{dQ}{dt} = R(t) - (k + \lambda)Q \qquad (5\text{-}17)$$

where

Q = the contaminant inventory in the leach pit (Ci or mg of contaminant),

$R(t)$ = the contaminant input rate (Ci or mg per year),

k = the removal or leach rate constant (1/y), and

λ = the decay rate constant (1/y).

The following assumptions were made pertaining to this model:

- The rate of contaminant discharge to the pit was proportional to the effluent flow rates
- No contaminants left the leach pit after liquid discharges were ceased to the time the pit was excavated (because of no driving force for migration, the contaminants were inside a building with poured concrete walls and a concrete roof)
- No contaminants left the leach pit after the sludge was removed (the sludge, walls, and roof were removed and a bentonite layer was installed over the bedrock to inhibit the infiltration of water as the driving force)
- The majority of the contamination was removed during sludge excavation

If we assume that no contaminants were removed (leached) from the pit between the years 1973 to 1993 as stated in the second bullet, then the inventory of contaminants (Q) in 1973 can be determined by simply decay-correcting the concentrations measured in 1991 back to 1973. Knowing the inventory in the pit in 1973 allows for the determination of R(t) or the contaminant release rate. For input into the GWSCREEN pond model, the release rate was averaged over the entire time of operation of the pit. The removal or leach rate constant (k) describes the fraction of the inventory in the pit that is leached to the subsurface. Equation 1 was solved for a total disposal mass of 1 unit using a Runge-Kutta solving routine described in Press et al. (1992). The function R(t) was the fraction of the total inventory disposed in the waste pit for a given year. Fractions were proportional to the liquid effluent discharge rate to the pit (Figure 5-5).

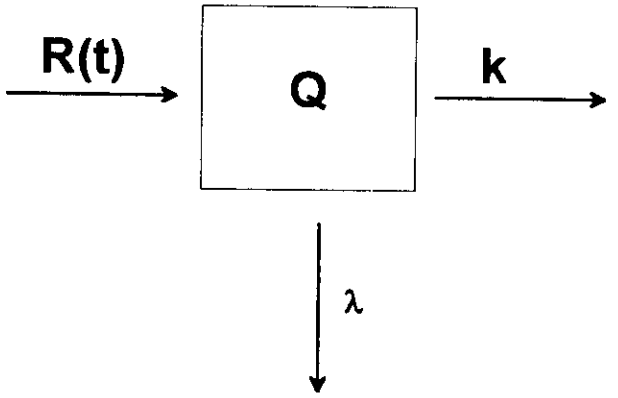

Figure 5-4. Conceptual model for EBR-II Leach Pit operation, 1958–1973.

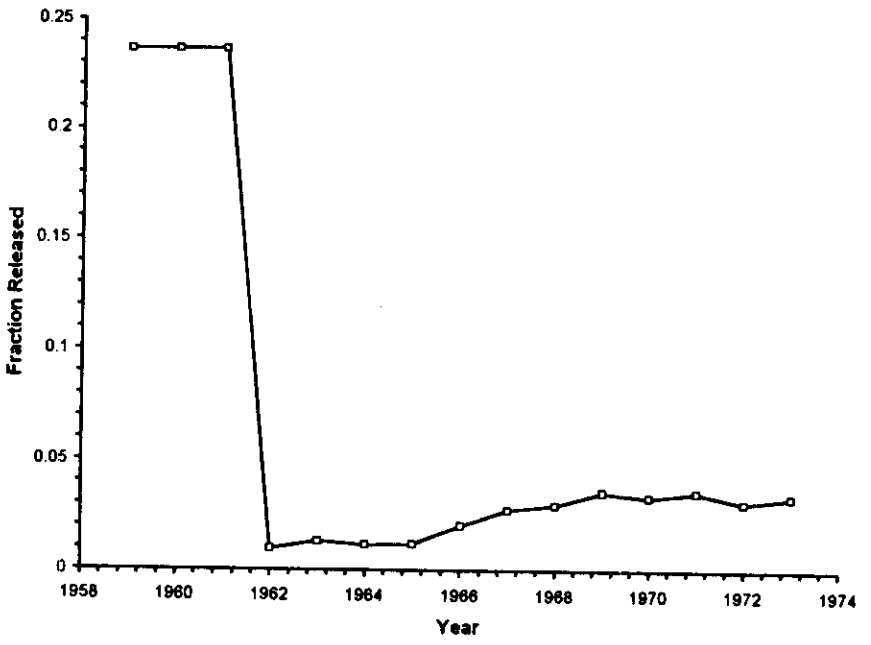

Figure 5-5. Fraction of contaminant inventory released to the leach pit as a function of time for the operation period of the pit (1958-73).

The total mass or activity released to the pond can now be found by a simple ratio as given by Equation (2).

$$I = \frac{I_{\text{mod}}\ Q_{meas}(te)}{Q_{\text{mod}}(te)} \tag{5-18}$$

where

I = the total mass or activity disposed in the pit (mg or Ci)

I_{mod} = the modeled mass or activity disposed in the pit (1 mg or 1 Ci)

Q_{meas}(te) = the measured inventory in the pit at the end of operation (1973)

Q_{mod}(te) = the modeled inventory in the pit at the end of operation (1973).

The quantity I was averaged over the 15 years of pit operation and input into GWSCREEN as the contaminant input rate. The liquid effluent flow rate used in the GWSCREEN calculation was the average discharge rate over the 15 years of pit operation (564 m^3/y). The leach rate constant was calculated using the equation

$$k = \frac{P}{\theta\left(1+\frac{K_d\rho}{\theta}\right)T} \tag{5-19}$$

where

θ = the volumetric moisture content (0.41),

T = contaminated thickness (0.11 m),

K_d = the contaminant specific sorption coefficient (mL/g),

P = the infiltration rate (8.7 m/y), and

ρ = bulk density (1.5 g/cm^3).

Contaminant thickness was assumed to be equal to the thickness of sludge calculated to be at the bottom of the pit. This value was calculated by dividing the total volume of sludge removed (7.34 m^3) by the area of the pit (69 m^2). Hydraulic properties of the contaminated volume were assumed to be the same as for the surface soil described in Table 5-25. The volumetric moisture content was based on the moisture characteristic curve for the surface soils. Results of the calculation are presented in Table 5-26.

Table 5-25. Results of EBR Leach Pit source term calclations

Contaminant	Half-life (years)	Kd (mL/g)	Leach rate (1/yr)	Measured inventory 1991 (mg or Ci)	Measured inventory 1973 (decay corrected) (mg or Ci)	Modeled Inventory 1973 (mg or Ci)	Total mass or activity input (mg or Ci)	Average mass release rate (mg or Ci/y)
Antimony	infinity	5.0	9.39E+00	2.79E+05	2.79E+05	3.36E-03	8.30E+07	5.54E+06
Arsenic	infinity	3.0	1.51E+01	2.92E+05	2.92E+05	2.03E-03	1.44E+08	9.57E+06
Barium	infinity	50.0	9.85E-01	2.48E+06	2.48E+06	3.30E-02	7.51E+07	5.00E+06
Beryllium	infinity	250.0	1.98E-01	2.78E+06	2.78E+06	1.85E-01	1.50E+07	1.00E+06
Cadmium	infinity	6.0	7.89E+00	2.85E+05	2.85E+05	4.03E-03	7.07E+07	4.72E+06
Chromium	infinity	1.2	3.36E+01	2.27E+07	2.27E+07	8.36E-04	2.72E+10	1.81E+09
Cobalt	infinity	10.0	4.82E+00	1.09E-05	1.09E+05	6.70E-03	1.62E+07	1.08E+06
Copper	infinity	20.0	2.44E+00	1.04E-08	1.04E+08	1.33E-02	7.84E+09	5.23E+08
Cyanide	infinity	0.0	1.80E+02	1.65E+05	1.65E+05	8.50E-05	1.94E+09	1.29E+08
Lead	infinity	100.0	4.94E-01	1.85E+06	1.85E+06	6.45E-02	2.87E+07	1.91E+06
Manganese	infinity	50.0	9.85E-01	3.25E+06	3.25E+06	3.30E-02	9.86E+07	6.57E+06
Mercury	infinity	100.0	4.94E-01	3.47E+06	3.47E+06	6.45E-02	5.37E+07	3.58E+06
Nickel	infinity	100.0	4.94E-01	5.37E+05	5.37E+05	6.45E-02	8.32E+06	5.55E+05
Selenium	infinity	4.0	1.16E+01	1.65E+03	1.65E+03	2.70E-03	6.12E+05	4.08E+04
Silver	infinity	90.0	5.49E-01	1.44E+05	1.44E+05	6.00E-02	2.40E+06	1.60E+05
Sulfate	infinity	0.0	1.81E+02	5.15E+05	5.15E+05	8.50E-05	6.05E+09	4.04E+08
Thallium	infinity	0.0	1.81E+02	1.58E+05	1.58E+05	8.50E-05	1.86E+09	1.24E+08
Vanadium	infinity	1000.0	4.95E-02	4.39E+05	4.39E+05	5.99E-01	7.34E+05	4.89E+04
Zinc	infinity	2.5	1.79E+01	1.48E+07	1.48E+07	1.70E-03	8.73E+09	5.82E+08
HpCDD	infinity	0.0	1.81E+02	6.56E+02	6.56E+02	8.50E-05	7.71E+06	5.14E+05
HpCDF	infinity	0.0	1.81E+02	1.09E+02	1.09E+02	8.50E-05	1.28E+06	8.55E+04
HxCDD	infinity	0.0	1.81E+02	3.57E+02	3.57E+02	8.50E-05	4.20E+06	2.80E+05
HxCDF	infinity	0.0	1.81E+02	1.00E+02	1.00E+02	8.50E-05	1.18E+06	7.86E+04
OCDD	infinity	0.0	1.81E+02	3.11E+03	3.11E+03	8.50E-05	3.66E+07	2.44E+06
OCDF	infinity	0.0	1.81E+02	5.80E+01	5.80E+01	8.50E-05	6.82E+05	4.55E+04
PeCDD	infinity	0.0	1.81E+02	6.16E+01	6.16E+01	8.50E-05	7.25E+05	4.84E+04
PeCDF	infinity	0.0	1.81E+02	1.74E+01	1.74E+01	8.50E-05	2.05E+05	1.37E+04
TCDD-TOT	infinity	0.0	1.81E+02	2.42E+00	2.42E+00	8.50E-05	2.85E+04	1.90E+03
TCDF	infinity	0.0	1.81E+02	5.50E+00	5.50E+00	8.50E-05	6.48E+04	4.32E+03
Am-241	4.32E+02	340.0	1.46E-01	7.16E-06	7.17E-06	2.57E-01	2.79E-05	1.86E-06
Co-60	5.75E+00	10.0	4.82E+00	1.18E-03	1.33E-03	6.50E-03	2.05E-01	1.36E-02
Cs-134	2.06E+00	500.0	9.90E-02	1.33E-05	1.86E-05	7.50E-02	2.48E-04	1.66E-05
Cs-137	3.00E+01	500.0	9.90E-02	1.99E-01	2.03E-01	3.11E-01	6.54E-01	4.36E-02
I-129	1.57E+07	0.1	1.53E+02	1.37E-03	1.37E-03	1.10E-04	1.24E+01	8.27E-01
Np-237	2.10E+06	5.0	9.39E+00	2.19E-03	2.19E-03	3.36E-03	6.51E-01	4.34E-02
Pu-238	8.77E+01	22.0	2.22E+00	2.31E-06	2.33E-06	1.46E-02	1.60E-04	1.06E-05
Pu-239	2.41E+04	22.0	2.22E+00	3.15E-05	3.15E-05	1.46E-02	2.16E-03	1.44E-04
Sr-90	2.91E+01	24.0	2.04E+00	1.01E-02	1.03E-02	1.58E-02	6.53E-01	4.36E-02
U-234	2.45E+05	6.0	7.89E+00	2.85E-04	2.85E-04	4.03E-03	7.08E-02	4.72E-03
U-235	7.04E+11	6.0	7.89E+00	1.49E-05	1.49E-05	4.03E-03	3.70E-03	2.46E-04
U-238	4.47E+09	6.0	7.89E+00	2.61E-05	2.61E-05	4.03E-03	6.47E-03	4.32E-04

It is likely that the hydraulic properties of the soil at the base of the pit changed with time as evidenced by the presence of a sludge layer. However, at this point, it is difficult to estimate these changes without additional characterization. The ultimate validity of the model depends on the results of the sampling data in the interbeds below the leach pit. The model presented here was only used to arrive at an upper-bound estimate of the total mass or activity disposed in the pit and the fraction that was leached. It should be understood that the uncertainty in such estimates are large to say the least. But, without better more exact release records for the operational history of the EBR-II Leach Pit, it presents the most probable releases based on the known sludge sampling results.

5.8 Groundwater Results

5.8.1 Maximum Concentrations

Maximum concentrations and time of maximum concentration in the aquifer are presented in Tables 5-27 through 5-30. The time of maximum is the number of years from the present (1997) except for ANL-08 EBR-II Leach Pit. The time of maximum for this release site is the year 1958, the startup time of the Leach Pit.

Table 5-26. Maximum groundwater concentration and time of maximum for ANL-09—Canal, ANL-09—Mound, and ANL-01—Ditch B. Time of maximum is the number of years from 1997.

	ANL-09—Ditch		ANL-09—Mound		ANL-01—Ditch B #1		ANL-01—Ditch B #2	
	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)
Cs-137	0.0E+00	n/a	0.0E+00	n/a				
Cm-244			0.0E+00	n/a				
Co-60	1.7E-17	65	0.0E+00	n/a				
U-238 (parent)			3.2E-13	1410				
U-234 (progeny)			1.3E-15					
Th-230 (progeny)			5.0E-19					
Ra-226 (progeny)			1.7E-18					
Pb-210 (progeny)			8.3E-20					
2, 4-D					6.0E-05	50	2.1E-04	60
Arsenic	8.2E-02	22			3.4E-04	730	8.4E-04	89
bis (2-Ethylhexyl) phalate					9.5E-03	4129	2.3E-06	5010
Chromium					1.2E-01	322	3.1E-01	390
Copper					9.1E-04	4582	2.2E-03	5560
Lead					3.4E-05	22710	8.2E-05	27,600
Methylene Chloride					1.3E-05	56	4.3E-05	67
Selenium					7.6E-05	953	1.8E-04	1160
Zinc					2.8E-02	3675	6.6E-02	4460

Table 5-27. Maximum groundwater concentration and time of maximum for ANL-01—Ditch C, ANL-01—Ditch A, and ANL-01—Industrial Waste Pond. Time of maximum is the number of years from 1997.

	ANL-01—Ditch C		ANL-01—Ditch A #1		ANL-01—Ditch A #2		ANL-01—IWP	
Contaminant	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximun Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximum Concentraiton (mg or Ci/L)	Time of Maximum (Years)
Cs-137							1.0E-16	271
Cm-244							4.2E-14	7.4
Co-60							1.1E-14	6.6
Sr-90			2.2E-15	211	3.1E-17	301	2.2E-13	18
U-238 (parent)	6.5E-12	53.4	1.4E-12	66	1.8E-13	96		
U-234 (progeny)	9.9E-16	53.4	2.6E-16	66	4.9E-17	96		
Th-230 (progeny)	1.5E-19	53.4	4.8E-20	66	1.3E-20	96		
Ra-226 (progeny)	2.2E-20	53.4	9.0E-21	66	3.5E-21	96		
Pb-210 (progeny)	3.5E-22	53.4	1.6E-22	66	8.1E-23	96		
2, 4,5-Tp (Silvex)							1.1E-02	14
Arsenic	4.6E-03	28	5.6E-03	35	7.2E-04	50	2.5E-02	5.4
Cadmium							2.0E-03	11
Chloroform	6.8E-05	3.8					1.4E-04	0.5
Chromium			8.9E-02	16	1.2E-02	23	201	1
Copper			2.6E-03	212	3.3E-04	308	1.2	11
Cyanide	7.4E-02	2.7	3.0E-03	3.3	9.1E-04	4.6		
Fluoride	9.8E-02	2.7					0.16	0.4
Methylene Chloride	1.9E-03	2.9					5.2E-03	0.4
O-Phosphate	2.4E-01	2.6					8.0E-02	0.2
Selenium	7.8E-04	37					1.5E-01	2.5
Silver							3.0E-01	48
Sulfate	8.7E+00	2.6					58	0.4
Thallium	1.7E-05	2.6	1.2E-05	1050	1.5E-06	1510		
Zinc			2.3E-02	170	2.9E-03	248	27	9

Table 5-28. Maximum groundwater concentration and time of maximum for ANL-01A—MCTBD, ANL-35, and ANL-53. Time of maximum is the number of years from 1997.

	ANL-01A—MCTBD #1		ANL-01A—MCTBD #2		ANL-35		ANL-53	
Contaminant	Maximum Concentration (mg or Ci/L)	Time of Maxmimum (Years)	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)	Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)
Cs-137					4.8E-16	147		
Co-60					1.1E-14	1.9		
H-3					1.4E-11	0.1		
U-238 (parent)	1.0E-12	56	1.0E-12	56				
U-234 (progeny)	1.7E-16	56	1.7E-16	56				
Th-230 (progeny)	2.6E-20	56	2.6E-20	56				
Ra-226 (progeny)	4.1E-21	56	4.1E-21	56				
Pb-210 (progeny)	6.5E-23	56	6.5E-23	56				
2, 4-D					1.8E-03	0.1		
Acetonitrile					1.5E-03	0.1		
Antimony	8.6E-03	47	8.5E-03	47				
Arsenic	1.3E-03	29	1.2E-02	29				
bis (2-Ethylhexyl) phalate					7.0E-05	5.4		
Chloroform					1.9E-05	0.2		
Chromium	2.2E-01	13	2.2E-01	13	2.0	0.5	2.3E-02	428
Copper	2.5E-02	178	2.3E-02	178	5.8E-02	6		
Cyanide	1.6E-03	2.8	1.6E-03	2.8	5.8E-02	0.1		
Fluoride					9.5E-02	0.1		
HpCDD					8.0E-08	0.1		
HpCDF					8.0E-08	0.1		
HxCDF					8.0E-08	0.1		
Lead	2.9E-03	870	2.6E-03	870			7.5E-04	30200
Mercury	1.3E-04	870	1.2E-04	870				
Methylene Chloride	7.2E-05	3	7.2E-05	3	5.3E-03	0.1		
Nitrate					4.4E-01	0.1		
O-Phosphate					4.9E-02	0.1		
OCDD					2.0E-07	0.1		
OCDF					1.2E-08	0.1		
PeCDD					4.4E-09	0.1		
Selenium	5.4E-04	38	5.4E-04	38				
Silver								
Sulfate					2.8E+00	0.1		
Zinc	8.7E-02	143	8.2E-02	143	2.2E-01	4.8		

Table 5-29. Maximum groundwater concentration and time of maximum for ANL-08—Sludge. Time of maximum is the number of years from 1958.

Contaminant (Parent)	Progeny	ANL-08—Sludge Maximum Concentration (mg or Ci/L)	ANL-08—Sludge Time of Maximum (Years)
Am-241		1.8E-15	1189
	Np-237	1.4E-16	1189
	U-233	2.4E-20	1189
	Th-229	9.9E-22	1189
Cs-134		0.0E+00	n/a
Cs-137		6.1E-28	1740
Co-60		5.9E-12	51
I-129		6.5E-08	4
Np-237		3.3E-09	33
	U-233	4.0E-13	33
	Th-229	3.9E-17	33
Pu-238		3.2E-13	92
	U-234	4.4E-16	92
	Th-230	1.3E-19	92
	Ra-226	3.5E-20	92
	Pb-210	8.0E-22	92
Pu-239		8.3E-12	92
	U-235	2.7E-21	92
	Pa-231	1.6E-25	92
	Ac-227	8.6E-26	92
Sr-90		4.4E-10	85
U-234		3.6E-10	37
	Th-230	7.3E-14	37
	Ra-226	1.2E-14	37
	Pb-210	1.7E-16	37
U-235		1.9E-11	37
	Pa-231	8.8E-16	37

Contaminant (Parent)	Progeny	ANL-08—Sludge Maximum Concentration (mg or Ci/L)	ANL-08—Sludge Time of Maximum (Years)
	Ac-227	3.6E-16	37
U-238		3.3E-11	37
	U-234	3.4E-15	37
	Th-230	3.5E-19	37
	Ra-226	3.7E-20	37
	Pb-210	4.3E-22	37
Antimony		4.3E-01	33
Arsenic		7.5E-01	26
Barium		2.0E-01	189
Beryllium		9.7E-03	879
Cadmium		3.6E-01	37
Chromium		1.4E+02	18
Cobalt		7.6E-02	51
Copper		3.1E+01	85
Cyanide		9.8E+00	16
HpCDD		3.9E-02	9
HpCDF		6.5E-03	9
HxCDF		6.0E-03	9
Lead		4.9E-02	361
Maganese		2.7E-01	189
Mercury		9.2E-02	361
Nickel		1.4E-02	361
OCDD		1.9E-01	9
OCDF		3.5E-03	9
PeCDD		3.7E-03	9
Selenium		3.2E-03	30
Silver		4.4E-03	327
Sulfate		3.1E+01	9

Contaminant (Parent)	Progeny	ANL-08—Sludge Maximum Concentration (mg or Ci/L)	Time of Maximum (Years)
TCDF		3.3E-04	9
Thallium		3.2E+00	361
Vanadium		5.2E-05	3470
Zinc		3.7E+01	71

The 0–1,000 year (1997–2997) maximum contaminant concentrations that exceeded the risk-based concentration (Table 5-11 through 5-22) are identified in Table 5-30. Arsenic and Chromium were identified as the primary risk drivers. These contaminants were present at detectable levels at many of the sites and the ratio of their maximum concentration to risk-based concentration exceeded 1000. That is, their maximum concentration was 1000 times the risk-based concentration. The EBR-II leach pit (ANL-08) had the greatest number of contaminants that exceeded the risk-based concentration. In addition to arsenic and chromium, thallium and ^{90}Sr were identified as contaminants of concern from the leach pit.

Table 5-30 Ratio of 0–1000 year maximum concentration to the risk-based concentration for contaminants of concern.

Contaminant	ANL-09 Ditch	ANL-01 Ditch B	ANL-01 Ditch C	ANL-01 Ditch A	ANL-01 IWP	ANL-01A	ANL-08	ANL-35
Co-60 (Ci/L)							2.36E+00	
Sr-90 (Ci/L)							5.12E+02	
U-238 (Ci/L)			8.47E+00	1.83E+00		1.35E+00	4.25E+01	
Antimony							2.83E+01	
Arsenic	1.82E+03	1.86E+01	1.02E+02	1.24E+02	5.51E+02	2.78E+02	1.67E+04	
Cadmium							1.98E+01	
Chromium		1.72E+00			1.12E+03	1.24E+00	8.00E+02	1.10E+01
Copper							2.21E+01	
Cyanide							1.34E+01	
Lead							3.26E+00	
Mercury							8.33E+00	
Silver					1.65E+00			
Thallium							1.06E+03	
Zinc					2.41E+00		3.34E+00	

Concentration as a function of time was calculated at the receptor locations identified in Figure 5-3 for arsenic and. chromium. Concentrations were plotted as a function of time at four of these receptor

locations (Figures 5-6a and Figure 5-6b). Concentrations include contributions from all potential sources; However, the EBR-II Leach Pit (ANL-08), the Industrial Waste Pond (ANL-01), and the interceptor canal (ANL-09) were the primary sources of arsenic and chromium to the groundwater. Receptor 1 had the highest arsenic concentrations and was driven primarily by the interceptor canal source. The leach pit had the highest maximum concentration (0.75 mg/L, see Table 5-29), however, the maximum was predicted to have occurred in 1985 (27 years from 1958), which is prior to the time frame of interest. Chromium concentrations (Figure 5-6b) showed receptor 3 and 3 to have the highest concentrations. The leach pit and industrial waste pond were the primary sources of chromium to the groundwater, however the peak fluxes from these two sources did not overlap.

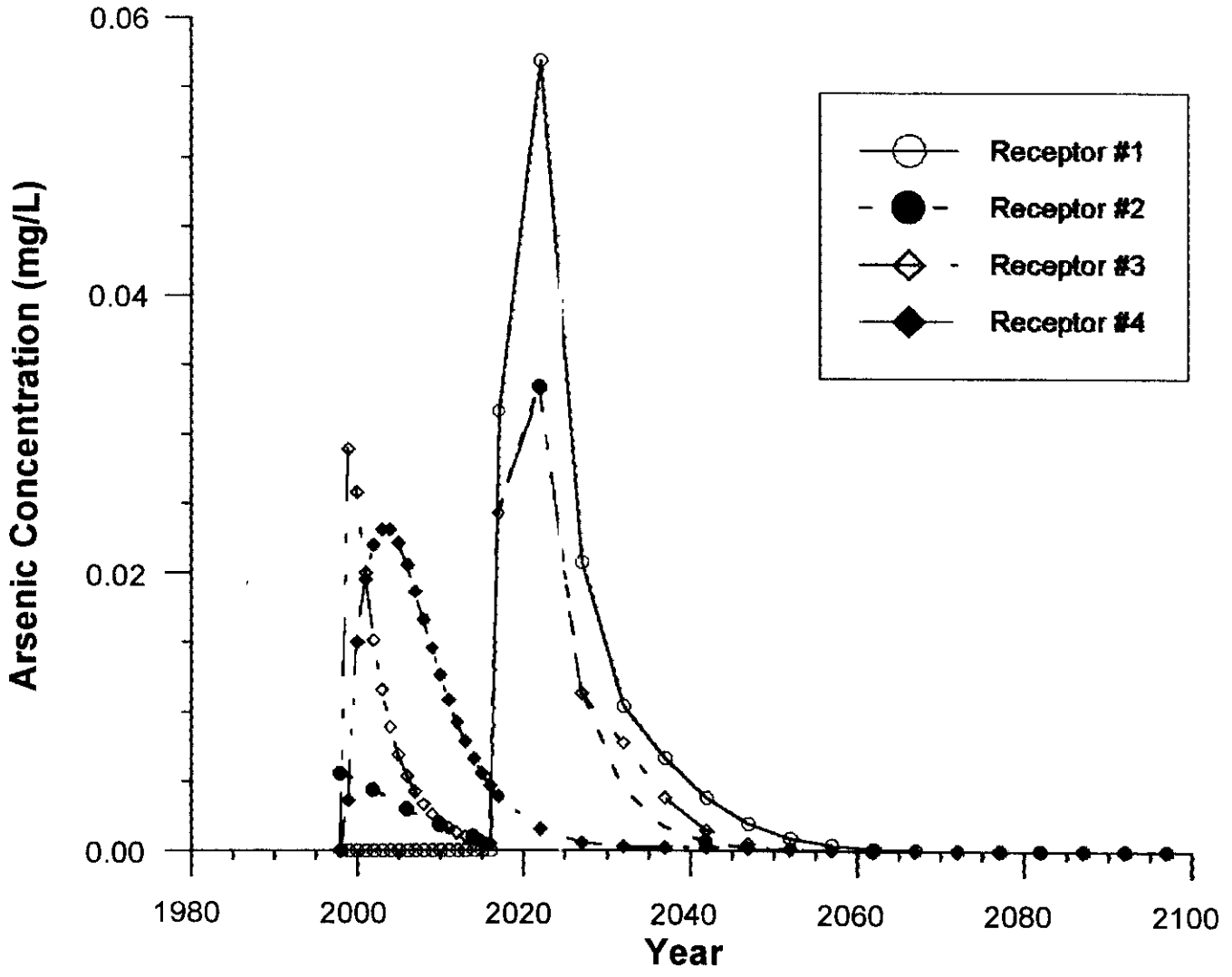

Figure 5-6a. Concentration as a function of time for arsenic at four locations downgradient from ANL-W (see Figure 5-3).

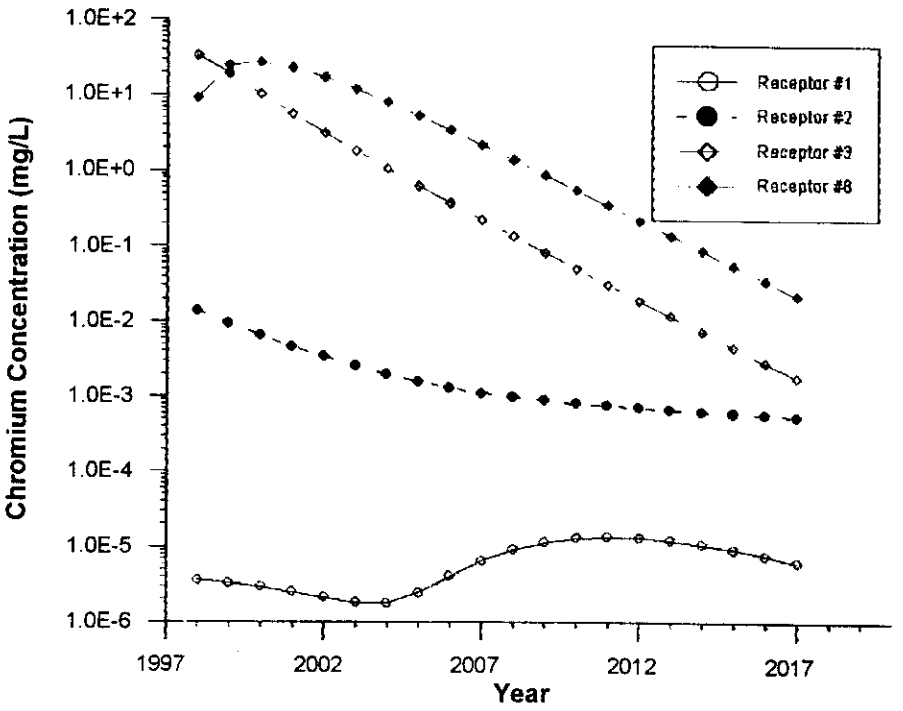

Figure 5-6b. Concentration as a function of time for chromium at four locations downgradient from ANL-W (see Figure 5-3).

5.8.2 100-year Concentrations for Selected Contaminants

Isopleth maps were generated for all retained sites that contained arsenic and chromium (Figure 5-7a and 5-7b). Concentrations in the model domain were calculated at 100 years from the present (2097). These isopleth maps indicate that the arsenic plume had moved about 500 m downgradient (west) from ANL-W and the chromium plume moved about 2 km west from ANL-W. The maximum 100-year arsenic concentration was 0.012 mg/L, and maximum 100-year chromium concentrations was 1.3 mg/L. Both these concentrations exceed the risk-based concentrations of 4.7E-05 mg/L for arsenic and 0.18 mg/L for chromium. Contaminant plumes were primarily influenced by contaminants in ANL-01—Industrial Waste Pond, ANL-08—EBR-II Leach Pit, and ANL-09—Interceptor Canal.

For contaminants that do not contribute carcinogenic risks greater than 1E-6 or a hazard index greater than 1 (i.e. their maximum concentration was less than the risk-based concentration), the maximum concentration value from the retained sites was used as the maximum groundwater concentration. Also, for those contaminants that were only identified in one release site at WAG 9 (i.e., no cumulative effect), the maximum groundwater concentration shown in Tables 5-23 through 5-29 is the combined maximum. This was the case for contaminants Alchlor 1260 and 2, 4, 5-TP (silvex) in release sites ANL-61A, and ANL-01 Industrial Waste Pond, respectively. However, since the contaminated soil in ANL-61A is scheduled for removal in the summer of 1997, the Alchlor 1260 groundwater concentration was not calculated. Until the removal, the Alchlor 1260 is not migrating very far (inches per year) because the K_d is relatively high (7,800 mL/g).

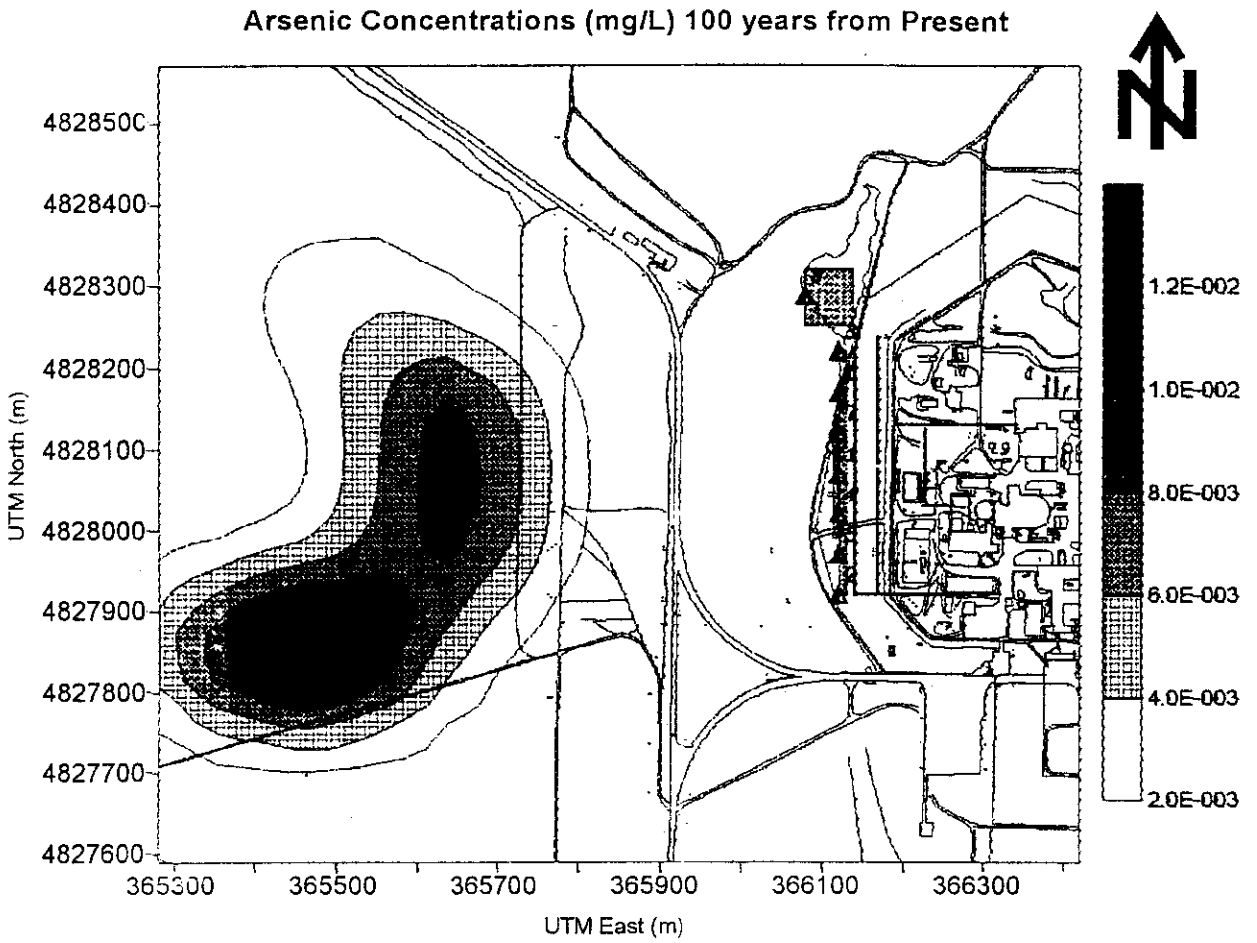

Figure 5-7a. Isopleth map of arsenic concentrations at 100 years (year 2097).

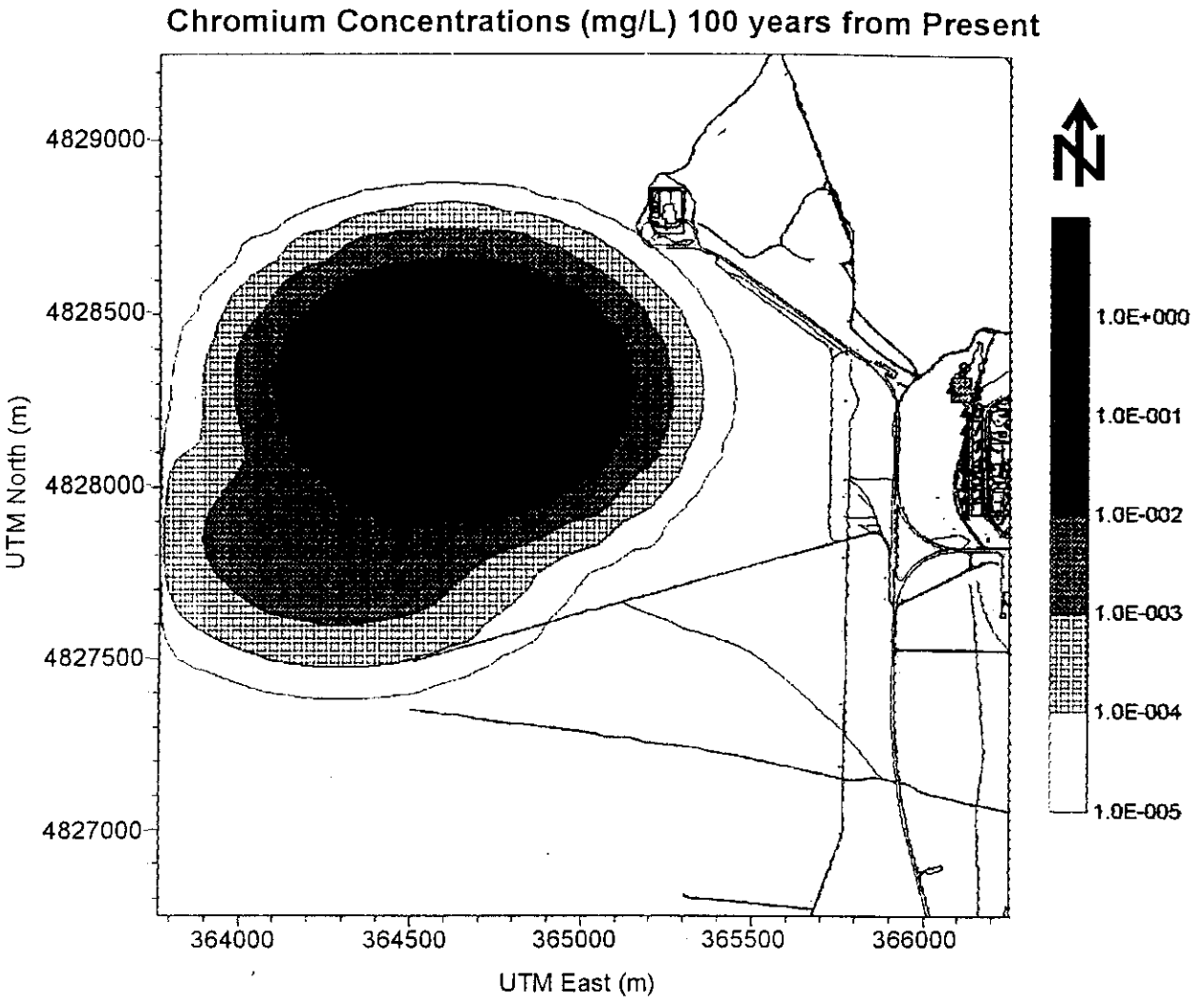

Figure 5-7b. Isopleth map of chromium concentrations at 100 years (year 2097).

5.9 Toxicity Assessment

This section provides the toxicity constants that will be used for risk characterization purposes and summarizes toxicological information for the WAG 9 radioactive and nonradioactive COPCs. For this assessment, and consistent with EPA's RAGS (EPA 1989), the toxicity information is summarized for two broad categories of potential effects: noncarcinogenic and carcinogenic effects. These two categories are selected because of the slightly differing methodologies for estimating potential health risks associated with exposures to carcinogens and noncarcinogens.

The toxicity constants used in the BRA are obtained from several sources. The primary source of information is EPA's Integrated Risk Information System (IRIS). IRIS contains only those toxicity constants that have been verified by EPA's Reference Dose or Carcinogen Risk Assessment Verification Endeavor (CRAVE) Work Groups. The IRIS database is updated monthly and supersedes all other sources of toxicity information. If the necessary data are not available in IRIS, EPA's HEAST (EPA 1994a) are used. The toxicity constant tables are published annually and updated approximately twice per year. HEAST contains a comprehensive listing of provisional risk assessment information that has been reviewed and accepted by individual EPA program offices, but has not had enough review to be recognized as high-quality, agency-wide accepted information (EPA 1994a).

5.9.1 Toxicity Assessment for Carcinogenic Effects

Potential carcinogenic risks are expressed as an estimated probability that an individual might develop cancer from lifetime exposure. This probability is based on projected intakes and chemical-specific dose-response data called carcinogenic SFs. Carcinogenic SFs and the estimated daily intake of a compound, averaged over a lifetime of exposure, are used to estimate the incremental risk that an individual exposed to that compound may develop cancer. This estimate is derived using the following equation:

$$\text{Risk} = \text{Intake} \times \text{SF} \qquad (5\text{-}20)$$

Risk = carcinogenic risk (unitless)

Intake = contaminant intake (mg/kg-day or pCi)

SF = slope factor [$(\text{mg/kg-day})^{-1}$ $(\text{pCi})^{-1}$].

There are two classes of potential carcinogens identified at WAG 9 release sites: chemical carcinogens and radionuclides. These two classes of carcinogens are discussed separately in the following sections.

5.9.1.1 Toxicity Assessment for Chemical Carcinogens. Evidence of chemical carcinogenicity originates primarily from two sources: (1) lifetime studies with laboratory animals, and (2) human (epidemiological) studies. For most chemical carcinogens, animal data from laboratory experiments represent the primary basis for the extrapolation. Major assumptions arise from the necessity of extrapolating experimental results: across species (i.e., from laboratory animals to humans); from high-dose regions (i.e., to which laboratory animals are exposed) to low-dose regions (i.e., levels to which

humans are likely to be exposed in the environment); and across routes of administration (i.e, inhalation versus ingestion). Federal regulatory agencies have traditionally estimated human cancer risks associated with exposure to chemical carcinogens on the administered-dose basis according to the following approach:

- The relationship between the administered dose and the incidence of cancer in animals is based on experimental animal bioassay results.

- The relationship between the administered dose and the incidence of cancer in the low-dose range is based on mathematical models.

- The dose-response relationship is assumed to be the same for both humans and animals, if the administered dose is measured in the proper units.

Effects from exposure to high (i.e., administered) doses are based on experimental animal bioassay results, while effects associated with exposure to low doses of a chemical are generally estimated from mathematical models.

For chemical carcinogens, EPA assumes that a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation and tumor induction. This mechanism for carcinogenesis is referred to as stochastic, which means that there is theoretically no level of exposure to a given chemical that does not pose a small, but finite, probability of generating a carcinogenic response.

Since risk at low exposure levels cannot be measured directly either in laboratory animals or human epidemiology studies, various mathematical models have been proposed to extrapolate from high to low doses (i.e., to estimate the dose-response relationship at low doses). The three most frequently used models are (1) the one-hit model, (2) the log-probit model, and (3) the multistage model (Armitage and Doll 1961). The one-hit model is based on the premise that a single molecule of a contaminant can be the single event that precipitates tumor induction (Cornfield 1977). In other words, there is some finite response associated with any exposure. The log-probit model assumes that a response is normally distributed with the logarithm of the dose (Mantel et al., 1971). This theory seems to have little scientific basis, although some physiological parameters are lognormally distributed. This model usually yields much lower potency estimates due to the implied threshold at lower doses.

Currently, regulatory decisions are based on the output of the linearized multistage model (EPA 1989). The basis of the linearized multistage model is that multiple events (versus the single-event paradigm of the one-hit model) may be needed to yield tumor induction. The linearized multistage model reflects the biological variability in tumor frequencies observed in animals or human studies (Crump et al. 1977). The dose-response relationship predicted by this model at low doses is essentially linear. Use of this model provides dose-response estimates intermediate between the one-hit and the log-probit models. It should be noted that the SFs calculated for nonradiological carcinogens using the multistage model represent the 95th percentile upper confidence limit on the probability of a carcinogenic response. Consequently, risk estimates based on these SFs are conservative estimates representing upper-bound estimates of risk where there is only a 5% probability that the actual risk is greater than the estimated risk.

Most models produce quantitatively similar results in the range of observable data, but yield estimates that can vary by three or four orders of magnitude at lower doses. Animal bioassay data are simply not adequate to determine whether any of the competing models are better than the others. Moreover, there is no evidence to indicate that the precision of low-dose risk estimates increases through the use of more sophisticated models. Thus, if a carcinogenic response occurs at the exposure level

studied, it is assumed that a similar response will occur at all lower doses, unless evidence to the contrary exists.

Uncertainties in the toxicity assessment for chemical carcinogens are dealt with by classifying each chemical into one of several groups, according to the weight of evidence from epidemiological studies and animal studies, as follows:

- Group A—Human Carcinogen (sufficient evidence of carcinogenicity in humans)
- Group B—Probable Human Carcinogen (B1-limited evidence of carcinogenicity in humans; B2—sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- Group C—Possible Human Carcinogen (limited evidence of carcinogenicity in the animals and inadequate or lack of human data)
- Group D—Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)
- Group E—Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies).

SFs for polyaromatic hydrocarbons (PAHs) are derived somewhat differently than SFs for other chemical carcinogens. Specifically, the methodology for estimating carcinogenic potencies of PAHs outlined in EPA's Environmental Criteria and Assessment Office's (ECAO's) *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons* (EPA 1993) was followed. The guidance uses a toxicity weighting factor approach to estimate the oral cancer SFs for several PAHs relative to the SF for benzo(a)pyrene (B(a)P). This methodology was developed because, while EPA has classified seven PAHs as Group B2-probable human carcinogens, data are sufficient to accurately measure dose-response (and by extension calculate SF) only for B(a)P.

To obtain an estimate of total carcinogenic risk resulting from modeled exposures to carcinogens at the site, cancer risks are summed across all exposure routes for all carcinogens. Cancer risks from exposure to multiple carcinogens across multiple pathways is assumed to be additive, based on EPA carcinogen risk assessment guidelines (EPA 1986a).

5.9.1.2 Toxicity Assessment for Radionuclides. An extensive body of literature exists that describes the health effects of radionuclides on humans and animals. Intensive research by national and international commissions has resulted in the establishment of widely accepted limits to which workers and the public may be exposed without clinically detectable effects. This literature has resulted in EPA classifying all radionuclides as Group A carcinogens since radionuclides emit ionizing radiation, which, at high doses, has been associated with increased cancer incidence in humans. Human epidemiological data collected from the survivors of the Hiroshima and Nagasaki bomb attacks form the basis for the most recent extrapolation put forth by the National Academy of Science (BEIR IV 1988). Conversely, for most nonradiological carcinogens, animal data from laboratory studies represent the primary basis for the extrapolation.

Another fundamental difference between the assessment of potential toxicity associated with exposure to radionuclide and nonradionuclide carcinogens is that SFs for radionuclides are typically best estimates (mean or median values rather than upper 95th percentile values). Furthermore, in the past, risk

factors for radionuclides have generally been based on fatalities (i.e., the number of people who actually died from cancer), while SFs for nonradiological carcinogens are based on incidence (i.e., the number of people who developed cancer). Finally, the SFs for radionuclides are expressed in different units, i.e., risk per picocurie $(pCi)^{-1}$ rather than $(mg/kg/day)^{-1}$.

These nonthreshold SFs account for the following: the amount of radionuclide transported into the bloodstream, the decay of radioactive progeny within the body, the distribution and retention of the radionuclide and its progeny (if any) in the body, the radiation dose delivered to specific organs and tissues, and the age and sex of the exposed individuals (EPA 1994a).

5.9.2 Toxicity Assessment for Noncarcinogenic Effects

Potential noncarcinogenic effects are evaluated by comparing daily intakes with chronic RfD developed by EPA. This section provides a definition of an RfD and discusses how it is applied in the BRA.

A chronic RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure that can be incurred during a lifetime, without an appreciable risk of a noncancer effect being incurred in human populations, including sensitive subgroups (EPA 1989). The RfD is based on the assumption that thresholds exist for noncarcinogenic toxic effects (e.g., liver or kidney damage). It is a benchmark dose operationally derived by the application of one or more order of magnitude uncertainty factors to doses thought to represent a lowest or no observed adverse effect level (LOAEL or NOAEL) in humans. Thus, there should be no adverse effects associated with chronic daily intakes below the RfD value. Conversely, if chronic daily intakes exceed this threshold level, there is a potential that some adverse noncarcinogenic health effects might be observed in exposed individuals.

RfDs or SFs have not been developed by EPA for the dermal exposure route. In the absence of these factors, the common practice has been to use the available toxicity measures for the oral route of exposure. This approach has been adopted in the BRA.

In evaluating the dermal pathway, EPA recommends expressing chemical intake as absorbed dose and adjusting the oral toxicity measures also to reflect absorbed dose (EPA 1989). In deriving such values, consistency is required between the type of dose that forms the basis of the oral toxicity and the type of dose that will be calculated by the dermal exposure models. Specifically, a distinction must be made between an administered dose or intake (i.e., the amount of chemical taken into the body) and the absorbed dose (i.e., the amount of chemical that crosses the body membranes and enters the bloodstream). Most of the toxicity measures available from EPA are expressed as administered dose (i.e., intake) rather than dose at the tissue level (i.e., absorbed dose). The adjustment of the oral toxicity measure can be accomplished only if sufficient data are available in the principal laboratory studies or on the oral absorption efficiency in the species on which the toxicity measures are based. EPA notes that exposure estimates for absorption efficiency should not be adjusted if the toxicity values are based on administered doses (EPA 1989).

For risk characterization purposes, potential health effects of chronic exposure to noncarcinogenic compounds will be assessed by calculating a hazard quotient (HQ) for each COPC. A HQ will be derived by dividing the estimated daily intake by a chemical-specific RfD as shown in this equation:

$$HQ = RfD/Intake \qquad (5\text{-}21)$$

where

HQ = hazard quotient (unitless)

RfD = reference dose (mg/kg-day or pCi)

Intake = contaminant intake (mg/kg-day or pCi).

A HQ greater than 1.0 indicates that exposure to a given contaminant (at the concentrations and for the duration and frequencies of exposure estimated in the exposure assessment) may cause adverse health effects in exposed populations. However, the level of concern associated with exposure to noncarcinogenic compounds does not increase linearly as HQ values exceed 1.0. In other words, HQ values do not represent a probability or a percentage. For example, an HQ of 10 does not indicate that adverse health effects are 10 times more likely to occur than an HQ value of 1.0. All one can conclude is that HQ values greater than 1.0 indicate that noncarcinogenic health impacts are possible and that the more an HQ value exceeds unity, the greater the concern about potential adverse health effects.

Consistent with RAGS, chemical-specific HQs are summed across exposure routes to calculate a Hazard Index (HI) for each COPC. Individual pathway HI values are then summed to determine a cumulative HI value for all exposure pathways and COPCs at each release site. This approach may result in a situation where a total HI value for a given release site may exceed unity even though none of the chemical-specific HQ values at the release site exceed unity.

5.9.3 Toxicity Profiles

A toxicity assessment was conducted to identify potential adverse effects and toxicity values for contaminants at WAG 9. A toxicity value is a numerical expression of a substance dose-response relationship that is used in the risk assessment.

The following types of toxicity values are used in risk assessment: (a) reference doses (RfDs) and reference concentrations (RfCs), which are used to evaluate noncarcinogenic effects, and (b) slope factors (SFs) and unit risk values, which are used to evaluate carcinogenic effects. EPAs *Integrated Risk Information System* (IRIS) database and the *Health Effects Assessment Summary Tables* (HEAST) provided the majority of the toxicity values for this assessment.

5.9.3.1 Radionuclides. The radionuclides evaluated for carcinogenic effects are listed in Table 5-31. EPA classifies all radionuclides as Group A carcinogens based on emissions of ionizing radiation and on the extensive weight of evidence provided by epidemiological studies of radiation-induced cancers in humans EPA (1995b). Target organs for radiation-induced cancers in humans can include the thyroid, breast, lung, blood (bone marrow), stomach, liver, small and large intestine, brain, bone, esophagus, bladder, pancreas, lymphatic tissues, skin, pharynx, uterus, ovary, and kidney (EPA 1991). As with chemical carcinogens, it is assumed that any dose of radiation can produce adverse effects and that no threshold exists for radiation carcinogenesis.

Slope factors used to evaluate carcinogenic effects for the radionuclides are obtained from EPA (1995b) and are shown in Table 5-31. Pathway specific slope factors are identified for ingestion, inhalation, and external exposure. A brief description of bodily effects of the radionuclides present at WAG 9 is presented below.

Table 5-31. Slope factors used in the evaluation of carcinogenic effects of radionuclides at WAG 9.

Radionuclide	Ingestion Sf[a] $(pCi)^{-1}$	Inhalation Sf[a] $(pCi)^{-1}$	External Exposure Sf[a] $[y/pCi/g]^{-1}$
Am-241	3.28E-10	3.85E-08	4.59E-09
Cm-244	2.11E-10	2.43E-08	2.07E-11
Co-60	1.89E-11	6.88E-11	9.76E-06
Cs-134	4.73E-11	2.89E-11	5.88E-06
Cs-137+D[b]	3.16E-11	1.91E-11	2.09E-06
H-3	7.15E-14	9.59E-14	0.00E+00
I-129	1.84E-10	1.22E-10	2.69E-09
Np-237+D[b]	3.00E-10	3.45E-08	4.62E-07
Pu-238	2.95E-10	2.74E-08	1.94E-11
Pu-239	3.16E-10	2.78E-08	1.26E-11
Sr-90+D[b]	5.59E-11	6.93E-11	0.00E+00
U-234	4.44E-11	1.40E-08	2.14E-11
U-235+D[b]	4.70E-11	1.30E-08	2.65E-07
U-238+D[b]	6.20E-11	1.24E-08	5.25E-08

a. Toxicity values from HEAST (EPA, 1995a).

b. Includes effects of its daughter products.

5.9.3.1.1 Americium—Data from animal studies have shown the absorption of americium from the gastrointestinal tract to be very low. Americium compounds are more rapidly cleared from the lung than are compounds of plutonium (ICRP 1978).

After inhalation, Am-241 resides more in the skeleton than in the lung (BEIR IV 1988). Thirty percent of Am-241 resides in the liver. Inhalation has been shown to induce lung tumors in rats (BEIR IV 1988).

5.9.3.1.2 Cesium/Barium—Irrespective of the mode of administration, Cs-137 is rapidly absorbed into the bloodstream and distributes throughout the active tissues of the body. Metabolically, Cs-137 behaves as an analog of potassium. Its distribution throughout the body and the energetic beta and gamma radiation from its decay daughter, Ba-137m, result in essentially whole-body irradiation (Amdur et al. 1991).

5.9.3.1.3 Cobalt—Organically complexed forms of cobalt are readily absorbed into the blood from the gastrointestinal tract, while oxides and hydroxides of cobalt are not (ICRP 1978).

Experiments have shown that a majority of the cobalt entering the body is retained in the liver (ICRP 1978). Treatment with Co-60 is used to kill cancer cells, but cobalt radiation can also affect normal cells and tissues in the body causing cancer. These radiation effects can include serious damage to the lungs, heart, intestines, blood cells, bone, and skin (Hall 1978).

5.9.3.1.4 Curium—Data from animal studies have shown the absorption of curium from the gastrointestinal tract to be very low. Greater absorption might be expected for complexed forms of curium and enhanced absorption has been reported in very young rats (ICRP 1978). As with americium, curium compounds, including the oxide, are more quickly lost from the lung than are the corresponding compounds of plutonium.

Tissues of interest with respect to potential health effects following intake of a transuranic element are lungs, liver, bone (bone marrow), and lymph nodes, and to a lesser degree thyroid gland, gonads, and kidney (BEIR IV 1988). By far the greatest emphasis has been placed on lungs and bone since these two tissues have been the predominant sites of neoplasia in experimental animals.

5.9.3.1.5 Hydrogen—Ingested tritiated water is assumed to completely and instantaneously absorbed from the gastrointestinal tract and to mix rapidly with the total body water so that, at all times following ingestion, the concentration in sweat, sputum, urine, blood, insensible perspiration and, expired water vapor is the same (ICRP 1978). Exposure to an atmosphere contaminated by tritiated water results in intake by both inhalation and by absorption through the intact skin.

When tritium-labeled organic compounds are ingested, a considerable fraction may be broken down in the gastrointestinal tract producing tritiated water. Organic compounds of tritium may also catabolize to tritiated water after they have crossed the gut. Many organic compounds of tritium are not very volatile under normal circumstances and the probability of their being inhaled as vapors is small (ICRP 1978).

5.9.3.1.6 Iodine—Iodine is absorbed rapidly and almost completely from the gastrointestinal tract, mainly from the small intestine, and the lungs. Approximately 30% of the iodine that enters the blood is retained in the thyroid (ICRP 1978). The iodine is eventually lost from the thyroid gland in the form of organic iodine and is retained in the remaining organs and tissues within the body. The biological half-life of iodine within the body is approximately 120 days (ICRP 1978).

5.9.3.1.7 Neptunium—Data from animal studies have shown the absorption of neptunium from the gastrointestinal tract to be very low. Experiments on rats indicate that neptunium is cleared from the lungs more rapidly than plutonium. Data on the distribution and retention of neptunium in the rat indicate that its metabolic behavior is rather similar to that of plutonium. However, there are some indications that in the skeleton, neptunium may distribute more like calcium than like plutonium (ICRP 1978).

Tissues of interest with respect to potential health effects following intake of a transuranic element are lungs, liver, bone (bone marrow), and lymph nodes, and to a lesser degree thyroid gland, gonads, and kidney (BEIR IV 1988). By far the greatest emphasis has been placed on lungs and bone since these two tissues have been the predominant sites of neoplasia in experimental animals.

5.9.3.1.8 Plutonium—After inhalation, plutonium may remain in the lungs but can move to the bones and liver (BEIR V 1990). It generally stays in the body for a very long time and continues to expose the surrounding tissues to radiation (ATSDR 1990b). This will eventually increase the chance of

developing cancer, but it will be a number of years before such cancer effects become apparent. Approximately 50% of the plutonium that enters the blood is retained in the bone and 30% in the liver with retention times of 20 to 50 years (BEIR IV 1988). Inhalation can cause lung tumors in rats, and dermal absorption is limited (BEIR IV 1988).

Plutonium absorption from the gastrointestinal tract appears to be limited but is increased with decreased iron and calcium levels (BEIR IV 1988). Data have been reported that indicate a much higher gastrointestinal absorption for certain compounds of plutonium that are unlikely to be encountered in occupational exposures, e.g., hexavalent plutonium compounds, citrates, and other organic complexes. Absorption is also increased in the very young (ICRP 1978).

5.9.3.1.9 Strontium—Strontium, as a metabolic analog of calcium, is readily absorbed from the gastrointestinal tract or the lungs into the bloodstream and is subsequently deposited in the bones. A single brief intake orally, intravenously, or by inhalation, results in a high incidence of tumors of bones and bone-related tissues (BEIR V 1990).

The major point of attack of strontium is the lung via inhalation. Animal studies have shown lung and possible liver damage after exposure to strontium (Sittig 1985).

5.9.3.1.10 Uranium—Uranium and its compounds are highly toxic. Studies have shown that on the order of 0.005 to 0.05% of an uranium compound is likely to be absorbed into the blood from the gastrointestinal tract (ICRP 1978). Also, soluble uranium compounds such as UF_6, UO_2F_2, and $UO_2(NO_3)_2$ are rapidly absorbed from the lung, and retention times for uranium in the body may range from 20 to 50 years.

The major target organs for uranium toxicity are the respiratory system, blood, liver, lymphatics, kidneys, skin, and bone marrow. Reports confirm that carcinogenicity is related to dose and exposure time. Cancer of the lung, bone, and lymphatic tissues have been reported for soluble compounds, whereas cancer of the lymphatic and blood-forming tissues has been reported for insoluble compounds (Sittig 1985).

5.9.3.2 Nonradionuclides. The following WAG 9 chemicals are evaluated for noncarcinogenic effects based on the availability of toxicity data needed for the risk calculations: acetone, acetonitrile, antimony, arsenic, bis(2-ethylhexyl)phthalate, 2-butanone, butylbenzylphthalate, cadmium, chloroform, chromium, cobalt, copper, cyanide, 2,4-D, diethylphthalate, di-n-butylphthalate, di-n-octylphthalate, fluoride, lead, mercury, methylene chloride, nickel, nitrate, selenium, silver, strontium, thallium, toluene, 2,4,5-TP, 1,1,1-trichloroethane, and zinc. There is no toxicity data for lead, however, the risk posed by lead will be calculated using the Integrated Exposure Uptake Biokinetic (IEUBK) model (EPA 1994).

Based on the examination of available toxicity data, the following chemicals are evaluated for carcinogenic effects: arsenic, bis(2-ethylhexyl)phthalate, cadmium, chloroform, chromium (VI), lead, and methylene chloride. Arsenic and chromium (VI) are classified as a human carcinogen (Group A, sufficient evidence of carcinogenicity in humans); cadmium is classified as a probable human carcinogen (Group B1, sufficient evidence of carcinogenicity in animals with insufficient evidence in humans); and bis(2-ethylhexyl)phthalate, chloroform, and methylene chloride as probable human carcinogens (Group B2, sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans). Lead, a Group B2 carcinogen, is identified as a contaminant in the waste inventory, but is only evaluated qualitatively since no carcinogenic toxicity data are available for it.

The following sections describe the potential toxic effects associated with the evaluated exposure routes, and the sources of the toxicity values used in the toxicity assessment. Some of the toxicity values are derived based on available toxicity information and approved by EPA Environmental Criteria and Assessment Office (ECAO). EPA-established toxicity values used in the evaluation of noncarcinogenic and carcinogenic effects of chemicals are shown in Tables 5-32 and 5-33, respectively. Uncertainty factors(UF) used in deriving the toxicity values are also given

5.9.3.2.1 Acetone—Acetone is moderately toxic by various routes. The critical effects of acetone are increased liver and kidney weights, and kidney toxicity (EPA 1996). Human systemic effects by inhalation include changes in carbohydrate metabolism, nasal effects, conjunctiva irritation, respiratory system effects, nausea and vomiting, and muscle weakness (Sax 1989). Human systemic effects as a result of ingestion include coma, kidney damage, and metabolic changes (Sax 1989). Acetone is a narcotic at high concentrations.

The oral RfD for acetone, 1E-01 mg/kg-d, is obtained from IRIS (EPA 1996). The uncertainty factor of 1,000 reflects a factor of 100 for inter- and intra-species extrapolation and 10 to extrapolate from subchronic to chronic exposure. The overall confidence in the oral RfD is low, based on an evaluation of database and studies in the database. The confidence of the database is low because a very limited number of studies are available, and no pertinent supporting studies were located. Acetone is not evaluated for carcinogenic effects because it is not classifiable as to human carcinogenicity.

5.9.3.2.2 Arsenic—Acute exposure to arsenic causes severe throat irritation, gastrintestinal disturbances, and muscle spasms. This is followed by vertigo, delirium, and coma. Facial edema may also be evident. Sensor loss and hematopoietic symptoms associated with acute exposure are usually reversible. Chronic exposure, either by ingestion or inhalation, is marked by malaise and fatigue. Changes in the skin include hyperkeratosis. Anemia and neuropathy, liver injury, and "blackfoot disease" also result from chronic exposure.

Arsenic is a known carcinogen in humans. Ingestion is associated with increased incidence of skin cancer; lung cancer results from inhalation.

5.9.3.2.3 Antimony—The critical effects of antimony are longevity, blood glucose, and cholesterol (EPA 1996). Health effects have been observed in humans and animals following inhalation exposure to several antimony compounds. It is absorbed slowly form the gastrointestinal tract, and many antimony compounds are gastrointestinal irritants (Arndur et al. 1991). The toxicological effects of antimony in humans following inhalation or ingestion are lung disease, increased blood pressure, abdominal distress, ulcers, dermatosis, and ocular irritation (ATDSR 1992a). No effects were found in humans after dermal exposure to antimony.

An increase in the number of spontaneous abortions was observed in women exposed to airborne antimony in the workplace. No overt developmental effects were observed in the children of these women (ATSDR 1992a).

The oral RfD for antimony, obtained from IRIS (EPA 1996), is 4E-04 mg/kg-d. The uncertainty factor of 1,000 reflects a factor of 10 for inter-species conversion, 10 to protect sensitive individuals, and 10 because the effect level was a LOAEL and NOEL was established. The overall confidence in the oral RfD is low, based on an evaluation of database and studies in the database. Confidence in the chosen study is rated as low because only one species was used, only one dose level was used, no NOEL was determined, and gross pathology and histopathology were not well described. Confidence in the data base is low due to

Table 5-32. Toxicity values for noncarcinogens evaluated in the risk assessment for WAG 9.

Chemical	Critical Effect	Chronic oral RFD (mg/kg-d)	Chronic oral RFD UF	Chronic inhalation RfD (mg/kd-d)	Chronic inhalation RfC UF
Acetone	Increased liver and kidney weights, and kidney toxicity	1.0E-01	1000	ND	ND
Acetonitrile	Decreased red blood cell counts and hematocrit, and hepatic lesions	6.0E-03	3000	ND	ND
Antimony	Longevity, blood glucose, and cholesterol,	4.0E-04	1000	ND	ND
Bis(2-ethylhexyl)phthalate	Increased relative liver weight	2.0E-02	1000	ND	ND
2-Butanone	Decreased fetal birth weight	6.0E-01	3000	2.9E-01	3,000
Butylbenzylphthalate	Significantly increased liver-to-body weight and liver-to-brain weight ratios	2.0E-01	1000	ND	ND
Cadmium (water)	Significant protein in the urine	5.0E-04	10	ND	ND
Cadmium (food)	Significant protein in the urine	1.0E-03	10	ND	ND
Chloroform	Fatty cyst formation in liver	1.0E-02	1000	ND	ND
Chromium(III)	No critical effects reported	1.0E+00	1000	ND	ND
Chromium(VI)	No critical effects reported	5.0E-03	500	ND	ND
Cobalt	No critical effects reported	6.0E-02	ND	2.9E-04	ND
Copper		3.7E-02		ND	ND
Cyanide	Weight loss, thyroid effects and myelin degeneration	2.0E-02	100	ND	ND
2,4-D	Hematologic, hepatic and renal toxicity	1.0E-02	100	ND	ND
Diethylphthalate	Decreased growth rate, food consumption and altered organ weights	8.0E-01	1000	ND	ND
Di-n-butylphthalate	Increased mortality	1.0E-01	1000	ND	ND
Di-n-octylphthalate		2.0E-02		ND	ND
Fluoride		6.0E-02		ND	ND

Table 5-32. (continued).

Chemical	Critical Effect	Chronic oral RFD (mg/kg-d)	Chronic oral RFD UF	Chronic inhalation RfD (mg/kd-d)	Chronic inhalation RfC UF
Lead	No effects reported	ND	ND	ND	ND
Mercury (elemental)	Hand tremor, increases in memory disturbances, slight subjective and objective evidence of dysfunction of the autonomic nervous system	3.0E-04	ND	8.6E-05	30
Methylene Chloride	Liver toxicity	6.0E-02	100	8.6E-01	100
Nickel	Decreased body and organ weights	2.0E-02	300	ND	ND
Nitrate	Early clinical signs of hemoglobin in an oxidized state in the blood	1.6E+00	1	ND	ND
Selenium	Clinical selenosis	5.0E-03	3	ND	ND
Silver	Argyria	5.0E-03	3	ND	ND
Strontium		6.0E-01		ND	ND
Toluene	Changes in liver and kidney weights	2.0E-01	1000	ND	ND
2,4,5-TP	Histopathological changes in the liver	8.0E-03	100		
1,1,1-Trichloroethane		9.0E-02		2.9E-01	
Zinc	47% decrease in erythrocyte superoxide dismutase (ESOD) concentration in adult females after 10 weeks of zinc exposure	3.0E-01	3	ND	ND

ND = No data available

a. Values obtained from IRIS (EPA, 1996) except where noted.

b. Toxicity value obtained from HEAST (EPA, 1995a).

d. Oral RfD for methyl mercury.

e. MCL for nitrate is 10 mg/L as nitrogen.

lack of adequate oral exposure investigations. Antimony is not evaluated for carcinogenic effects because it is not classifiable as to human carcinogenicity.

Table 5-33. Toxicity values for carcinogens evaluated in the risk assessment for WAG 9.

Chemical	EPA weight-of-evidence	Oral Sf[a] $(mg/kg\text{-}d)^{-1}$	Inhalation SF[a] $(mg/kg\text{-}d)^{-1}$
Arsenic	A	1.8E+00	1.5E+01
Bis(2-ethylhexyl)phthalate	B1	1.4E-02	ND
Cadmium	B1	ND	6.3
Chloroform	B2	6.1E-03	8.1E-02
Chromium(VI)	A	ND	1.2E-02
Lead	B2	ND	ND
Methylene chloride	B2	7.5E-03	1.6E-03

ND = No toxicity data

a. Values obtained from IRIS (EPA, 1996) except where noted.

Antimony metal dust and fumes are absorbed from the lungs into the blood stream. Principal organs attacked include certain enzyme systems (protein and carbohydrate metabolism), heart, lungs, and the mucous membrane of the respiratory tract (Sittig, 1985).

Chronic oral poisoning presents symptoms of dry throat, nausea, headache, sleeplessness, loss of appetite, and dizziness. Liver and kidney degenerative changes are late manifestations (Sittig, 1985).

Antimony compounds are generally less toxic than antimony. Antimony trisulfide, however, has been reported to cause myocardial changes in man and experimental animals. Antimony trichloride and pentachloride are highly toxic and can irritate and corrode the skin. Antimony fluoride is extremely toxic, particularly to pulmonary tissue and skin (Sittig, 1985).

5.9.3.2.4 Bis(2-ethylhexyl)phthalate—Bis(2-ethylhexyl)phthalate (DEHP) is one or the most extremely studied phthalte esters for possible mutagenic, carcinogenic, and caranogenesis promoting potentials. Experimental studies have revealed caranogenic potential of DEHP in rats and mice. However, DEHP has failed to demonstrate a positive result in the short-term mutagenicity testing. Tetratogenic and reproductive effects have been observed in experimental animals. Chronic exposure to DEHP retarded growth and increased liver and kidney weights in animals. Reduced growth has been reported in cases involving chronic exposure to high concentrations of DEHP.

5.9.3.2.5 2-Butanone—The critical effect of 2-butanone is decreased fetal birth weight (EPA 1996). 2-Butanone is moderately toxic by ingestion, skin contact, and intraperitoneal routes. Human systemic effects by inhalation include conjunctiva irritation and irritation of the nose and pulmonary system (Sax 1989). It is also been shown that exposure to 2-butanone affects the peripheral nervous system and central nervous system (Sittig 1985). Experiments with animals have resulted in reproductive effects (Sax 1989).

The risk evaluation of 2-butanone is based on EPA-established toxicity values obtained from EPA (1996). The ingestion RfD for 2-butanone is 6.0E-01 mg/kg-d (EPA 1996). Three uncertainty factors of 10 each were applied when determining the RfD. One to account for inter-species extrapolation and intra-species variability (extrapolation to sensitive human populations); one to adjust for subchronic to chronic extrapolation since long-term effects in the females during the exposure period were not reported in the principal study; one for incompleteness of the data base, including a lack of both subchronic and chronic oral exposure studies for 2-butanone; and a modifying factor of three to account for the absence of data for a second rodent specie. As is usual practice, the application of four full areas of uncertainty generally results in a total uncertainty factor of 3,000, given the interrelationship among and overlap between the various areas of uncertainty described above. The assessment for methyl ethyl ketone (MEK) is based upon the strength of data supporting the use of the 2-butanol multi generation study and the concurrence of developmental effects for inhalation exposure to MEK and assumes that 2-butanol was not responsible for the fetal toxicity. There is a lack of data on the metabolism of 2-butanol and MEK over time thereby decreasing the confidence in the use of this surrogate approach. Therefore, confidence in the RfD is low.

The inhalation RfC for 2-butanone is 1.0E+00 mg/m^3 (EPA 1996). An uncertainty factor of 3,000 reflects factors of 10 to account for interspecies extrapolation, sensitive individuals, and incomplete data base including a lack of chronic and reproductive toxicity studies; and a modifying factor of three to address the lack of unequivocal data for respiratory tract (portal-of-entry) effects. The confidence in the RfD is low because of the lack of multi-generational studies and only one subchronic study in the database. 2-Butanone is not evaluated for carcinogenic effects because it is not classifiable as to human carcinogenicity.

5.9.3.2.6 Butylbenzylphthalate—Butylbenzylphthalate is considered a possible human carcinogen. It is classified as Group C chemical based on statistically significant increase in mononuclear cell leukemia in female rats; the response in male rats was inconclusive, and there was no such response in mice. This chemical significantly increased liver-to-body weight causes and liver-to-brain weight ratios in rats (NTP 1985).

5.9.3.2.7 Cadmium—The critical effect of cadmium is a significant amount of protein in the urine (EPA 1996). Ingestion of cadmium can cause adverse effects to the kidney, blood, liver at high concentrations, bone, testes in rats, gastrointestinal tract, and immune and cardiovascular systems. Symptoms from ingestion of cadmium include nausea, vomiting, salivation, abdominal pain, cramps, and diarrhea. In addition, it may cause reproductive and developmental effects (ATSDR 1993a). Because there is no evidence of cadmium causing cancer through ingestion, it is not considered a carcinogen by the EPA for this route.

Very little cadmium enters the body through the skin. A larger portion (5 to 8%) is absorbed by the intestinal tract (Amdur et al. 1991) and approximately 30 to 60% or cadmium which is inhaled is absorbed by the lungs (ATSDR 1993a) where the biological half life may be as long as 30 years (Amdur et al. 1991). Iron deficiency has been observed to increase cadmium uptake in humans and animals (ATSDR 1993a).

In animals, reproductive and developmental effects have been observed (ATSDR 1993a). Several factors affect the absorption of cadmium including age (ATSDR 1993a) and the placenta is a partial barrier. Cadmium is present in human milk. It is possible that cadmium exposure can increase the risk of prostate cancer (Sittig 1985).

Early symptoms from acute inhalation of cadmium fume include cough, pain in chest, sweating and chills, that progress to labored breathing, cough and generalized weakness (Sittig 1985). Kidney damage occurs from both inhalation and ingestion exposures and lung diseases (e.g., severe pulmonary irritation, bronchitis) from high inhalation exposures (ATSDR 1993a). Liver, testes, immune system, nervous system and the blood are damaged by cadmium exposures, and inhalation has been shown to cause cancer. Tumor locations are lung, trachea, and the bronchus (EPA 1996).

The EPA publishes two RfDs for cadmium: one for ingestion of food (1E-03 mg/kg-d) and the other for water ingestion (5E-04 mg/kg-d). Both of these values come from IRIS (EPA 1996). An uncertainty factor of 10 is used for both oral RfDs to account for sensitive individuals. Confidence in the oral toxicity values is high in that they are derived from a toxicokinetic model that uses data from several studies to calculate the absorption, distribution, metabolism and elimination of cadmium.

The EPA classifies cadmium as a B1 probable human carcinogen (i.e., sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) for inhalation (EPA 1996). The EPA inhalation unit risk for cadmium is 1.8E-03 $(\mu g/m^3)^{-1}$. This value was obtained from IRIS (EPA 1996). This value should not be used if the air concentration exceeds 6 $\mu g/mg^3$ because above this concentration the unit risk may not be appropriate (EPA 1996). The confidence in the inhalation unit risk is somewhat certain because it is derived from human data and the types of exposure are more realistic for environmental exposures (i.e., cadmium salts versus cadmium fumes and oxides).

5.9.3.2.8 Chromium—No critical effects for chromium have been reported (EPA 1996); however, acute exposure to chromium compounds can cause wheezing, coughing, headaches, labored breathing, pain and deep inhalation, fever, and loss of weight (Sittig 1985); whereas low levels of chromium III (i.e., 50 to 200 :g) is considered safe and adequate for normal body functions. Low levels of chromium cause weight loss, improper function of the nervous system, and a diabetic-like condition. Hexavalent chromium enters the body more readily than trivalent chromium, but is converted to trivalent chromium once it is in the body. Distribution in the body of chromium from inhalation by chromate workers is in the hilar lymph node, lungs, spleen, liver, kidney, and heart. Distribution of chromium after ingestion is in the liver, kidneys, and brain. Chromium may be transferred to the fetus through the placenta and to infants from breast milk. Very little chromium enters the body after skin contact unless the skin is damaged (ATSDR 1993b).

Both trivalent and hexavalent chromium can penetrate the skin to some extent, especially if the skin is damaged. Acute dermal exposure to hexavalent chromium causes skin ulcers/sores to develop on the area of contact (ATSDR 1993b) and treatment of scabies and facial carcinomas with chromium crystals and a salve containing chromium causes death. The rate of absorption for trivalent and hexavalent chromium varies depending on the chemical form of chromium (potassium dichromate, chromium sulfate) (ATSDR 1993b). The EPAs *Dermal Exposure Assessment: Principles and Applications* (EPA 1992) states that when the permeability coefficient for a chemical exceeds 1E-01 cm/hr in water then the dermal dose exceeds the ingested dose. There is no information on the absorption of chromium compounds from nonliquid medias.

Hexavalent chromium has been shown to be mutagenic in bacteria and yeast assays and can cause chromosomal effects (ATSDR 1993b).

Trivalent Chromium—this form of chromium is more commonly found in nature and is not as toxic as hexavalent chromium. It is this form of chromium that is believed to necessary for proper glucose functioning in the body and is, hence, an essential nutrient. However, swallowing large amounts may cause

severe health effects but no signs of carcinogenicity (ATSDR 1993b). Therefore, the EPA has established an oral RfD of 1E+00 mg/kg-d for insoluble salts (i.e , chromic oxide) based on NOEL in a feeding study (EPA 1996). The uncertainty factor for this RfD is 1,000 and it represents two 10-fold decreases in mg/kg bw/day dose that account for both the expected inter-human and inter-species variability to the toxicity of the chemical in lieu of specific data. There is an additional modifying factor of 10 because the effects observed in a 90-day study were not explicitly addressed in a 2-year study and there is a possibility that the NOEL listed may actually be a lowest-observed-adverse-effect level. There is low confidence in the data base because there is a lack of explicit detail on study protocol and results, lack of high-dose supporting data and the fact that no effects have been observed. Therefore, this RfD is considered to be conservative. The EPA is currently reviewing an inhalation reference concentration.

Hexavalent chromium—when taken orally, hexavalent chromium is absorbed in the tissue nine times higher than trivalent chromium, but no toxic effects have been observed (EPA 1996). Therefore, the EPA has established an oral reference dose based on a NOEL of soluble salts of hexavalent chromium of 5E-03 mg/kg-d (EPA 1996). The uncertainty factor for this RfD is 500. This is based on a factor of 10 for inter-human variability, another factor of 10 inter-species variability, and a modifying factor of five for less than lifetime exposure duration of the principal study. Confidence in the RfD is low because of the small numbers of animals and parameters studied, and the lack of toxic effects at the highest dose tested. Confidence in the database is low because studies are equally low quality and teratogenic and reproductive endpoints are not well studied (EPA 1996).

For the inhalation pathway, hexavalent chromium is classified by the EPA as a known human carcinogen (i.e., group A carcinogen) based on epidemiological studies of chromium-exposed workers (EPA 1996). In these studies, workers were exposed to both trivalent and hexavalent chromium. Because trivalent chromium has not been shown to cause cancer in animal studies, it has been concluded that hexavalent chromium is the carcinogen. The unit risk for inhalation obtained form IRIS (1996) is 1.2E-02 $(\mu g/mg^3)^{-1}$.

5.9.3.2.9 Cobalt—Cobalt is an essential component of vitamin B_{12} for the production of red blood cells and the prevention of pernicious anemia (Amdur et al. 1991). Cobalt salts are well absorbed after ingestion, and increased levels tend not to cause significant accumulation. Approximately 80% of the ingested cobalt is excreted in the urine with approximately 15% going out the feces. Milk and sweat are other mechanisms of excretion. Muscle contains the largest fraction in the body, followed by the liver, heart, and hair. The blood level is largely associated with red blood cells. Toxicity from therapeutic administration through ingestion results in vomiting, diarrhea, and a sensation of warmth. Therapeutic administration through injection causes flushing of the face, increased blood pressure, slowed respiration, giddiness, tinnitus and deafness due to nerve damage. Goiter is caused by high levels (i.e., 3 to 4 mg/kg) and alcohol can potentiate the effects of cobalt (Amdur et al. 1991). Beer fortified with cobalt causes a particular form of heart disease where massive amount of blood exit the sac around the heart (Calabrese 1991).

Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcomas at the site of injection, but there is no evidence of carcinogenicity from any other route of exposure (Amdur et al. 1991). Cobalt can adversely affect spermatogenesis and accessory sex organ function in addition to causing heart function to decrease (i.e., congestive heart failure) over the years because cobalt is antagonistic toward calcium, which is necessary for proper heart function. Respiratory irritation occurs in industrial exposures ranges from 0.002 to 0.01 mg/m^3.

5.9.3.2.10 Copper—Copper is not classified as to human carcinogenicity (EPA 1994b). Injection of various copper forms intramuscularly has shown no evidence of tumors in mice. A very low incidence of no injection site tumors were observed in rats, but these data are not conclusive. No increase in mutations were observed with the bacteria E. coli; however, DNA synthesis and chromosomal aberrations have been isolated viruses and rat liver cells, respectively (EPA 1994b).

Infants are sensitive to copper toxicity because their high copper levels and homeostatic mechanisms are not fully developed at birth. This is evidenced by hardening of the liver caused by formation of connective tissue followed by contraction after ingestion of milk contaminated with copper.

Copper salts act as irritants causing itching, erythema, and dermatitis (Sittig 1985). In the gastrointestinal (GI) tract, copper salts can cause nausea, vomiting, gastric pain, hemorrhagic gastritis, and diarrhea. This occurs at low concentrations (small levels) and because the stomach empties daily, it is not likely that this will progress in an industrial setting (Sittig 1985).

There are two types of genetically inherited inborn errors of copper metabolism: Wilson's disease and Menke's disease (Amdur et al. 1991). Wilson's disease causes excessive accumulation of copper in the liver, brain, kidneys and cornea, and the effects are clinical abnormalities of these organs. Menke's disease is a sex-linked trait characterized by kinky hair, failure to thrive (i.e., death before 3 years), severe mental retardation, and neurologic impairment (Amdur et al. 1991).

Copper is an essential metal for utilization of iron and several enzymes and iron-deficiency anemia in infancy is sometimes accompanied by copper deficiency (Amdur et al. 1991). Copper sulfate is used as an emetic, astringent, and anthelmintic. Copper sulfate mixed with lime is a fungicide. Most copper is stored in the liver and bone marrow where it is bound to metallothionein, and it is normally excreted in the bile. Brain levels tend to double from child to adult and ingestion of soft water significantly increases the amount of copper in the urine (Amdur et al. 1991).

5.9.3.2.11 Di-n-butylphthalate—No data on the carcinogenicity of di-n-butylphthalate are available. Di-n-butylphthalate exhibits low acute toxicity in laboratory animals. Rats injected with di-n-butylphthalate exhibited decreased birth weight, increased embryo mortality, and teratogenic effects (skeletal abnormalities) (Singh et al. 1972). Oral administration of 2,000 mg/kg-d for 9 days caused severe seminiferous tubular atrophy in rats and guinea pigs.

5.9.3.2.12 Di-n-octylphthalate—Although relatively little specific information concerning di-n-octylphthalate is available, the environmental transport and fate of this contaminant can be largely inferred from data for phthalate esters as a group. Di-n-octylphthalate probably hydrolyzes in surface waters, but at such a low rate that this process would not be significant under most conditions. Photolysis and oxidation do not appear to be important environmental fate processes (EPA 1985)

5.9.3.2.13 Lead—No critical effects of lead have been reported (EPA 1996). Many organs and systems are adversely affected by lead. The major target organs and systems are the central nervous system, the peripheral nerves, the kidney, the gastrointestinal system, and the blood system (Sittig 1985). Anemia is one of the early manifestations of lead poisoning. Other early effects of lead poisoning can include decreased physical fitness, fatigue, sleep disturbance, headache, aching bones and muscles, digestive symptoms, abdominal pains, and decreased appetite. The major central nervous system effects can include dullness, irritability, headaches, muscular tremors, inability to coordinate voluntary muscles, and loss of memory. The most sensitive effect for adults in the general population may be hypertension (Amdur et al. 1991).

Ingestion and inhalation of lead have the same effects on the human body. Large amounts of lead can result in severe convulsions, coma, delirium, and possibly death. A high incidence of residual damage is seen, similar to the ones following infections or traumatic damage or injury to sustained exposure to lead. In fact, most of the body burden of lead is in the bone (ATSDR 1990a). Lead effects in the peripheral nervous system is primarily manifested by weakness of the exterior muscles and sensory disturbances.

A major concern of lead exposure is behavioral effects, particularly in children. Exposure to lead can cause damage to the central nervous system or mental retardation or hearing impairment in children. Levels of exposure that may have little or no effect upon adults can produce important biochemical alterations in the growing child that may be expressed in altered neuropsychological behavior (Martin 1991).

Although the ability of lead to cause cancer in humans has not been shown, lead is classified by the EPA as a B2 probable human carcinogen (sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) through both ingestion and inhalation routes of exposure (EPA 1996). Lead classification is based on available evidence of cancer from animal studies. Rats exposed to lead in their diet had statistically increased incidence of kidney tumors (ATSDR 1990a).

Because laboratory animals fed lead in their diet throughout their lives have developed tumors, lead should be thought of as a probable cancer-causing substance in humans. There are some epidemiological studies of lead workers developing cancer, but the data are considered inadequate to demonstrate or refute the potential carcinogenicity of lead to humans.

It is possible that a couple may have trouble conceiving if the man has been exposed to high levels of lead because lead has been shown to affect sperm and damage other parts of the male reproductive system (ATSDR 1990a). Dermal absorption of inorganic lead compounds is reported to be much less significant than absorption by inhalation or oral routes of exposure (ATSDR 1990a).

There are no EPA-established toxicity values are available for lead. Therefore, a quantitative evaluation will be done using the IEUBK model (EPA 1994). The IEUBK model was developed for estimating risks from childhood lead exposure to soil and household dust that might be encountered at Superfund sites and RCRA corrective action facilities. If the lead concentrations are less than 400 mg/kg other models for lead exposures (construction worker or pregnant female) is not warranted.

5.9.3.2.14 Mercury—Mercury should only be present in metallic form at WAG 9. Each of the different forms of mercury have different effects on the human body. These effects are summarized in the following paragraphs.

Elemental mercury—the critical effects of elemental mercury include hand tremor, increases in memory disturbances, and slight subjective and objective evidence of dysfunction of the autonomic nervous system (EPA 1996). Harmful effects of elemental mercury include: coughing, chest pains, dyspnea, bronchitis, pneumonia, tremors, insomnia, irritability, indecision, headaches, fatigue, weakness, stomatitis, salivation, gastrointestinal disturbance, anorexia, weight loss, proteinuria, and irritation of the eyes and skin (Sittig 1985).

Several studies involving death in humans have been reported following acute exposure to high concentrations of metallic mercury vapor; death is attributed to a loss of respiratory function as a result of sever pulmonary tissue damage (ATSDR 1992b). In addition, workers chronically exposed to low concentrations of mercury exhibited double vision and acute exposure caused red burning eyes, and

conjunctivitis (ATSDR 1992b). In one case, a 13-year old boy was exposed to mercury vapors for two weeks and developed a thyroid enlargement (ATSDR 1992b), but studies on this effect in an occupational setting have not shown the same relationship.

Inorganic mercury—inorganic mercury is a primary irritant of skin and mucous membranes. It has been shown to cause allergic reactions on the skin (ATSDR 1992b). Acute poisoning due to mercury vapors affects the lungs primarily (e.g., acute interstitial pneumonitis, bronchitis, and bronchiolitis) (Sittig 1985). Mercury vapor has an affinity for the kidneys. Effects on the kidney include proteinuria (i.e., elevated serum proteins in the urine) (Amdur et al. 1991). The critical effect of inhalation of inorganic mercury is the same as that for elemental mercury (EPA 1996).

Ingestion exposure to lower levels varies from weakness, loss of appetite, loss of weight, insomnia, indigestion, diarrhea, metallic taste in mouth, increased salivation, and soreness of mouth or throat, to extreme irritability, excitability, anxiety, delirium with hallucinations, melancholia, and manic depressive psychosis. Acute exposures have been shown to cause severe abdominal cramps, bloody diarrhea, and suppression or urine. Corrosive ulceration, bleeding, and necrosis of the gastrointestinal tract are usually accompanied by shock and circulatory collapse. If the patient survives gastrointestinal damage, renal failure typically occurs in 24 hours. In general, chronic exposure causes increased excitability, tremors, and gingivitis (Amdur et al. 1991).

Spontaneous abortions have occurred in women who were exposed to mercury vapors. A significant increase in the number of spontaneous abortions also occurred when the fathers were exposed occupationally to mercury vapors. Metallic mercury vapors have been shown to absorb through the skin, but the majority of mercury vapor is absorbed by the lung through inhalation (ATSDR 1992b).

Organic mercury—the critical effect of organic mercury is developmental neurological abnormalities in human infants (EPA 1996). The local effect of dermal contact with organic mercury is dermatitis. Systemic effects are the central nervous system, primarily the brain. Severe poisoning can lead to irreversible brain damage resulting in loss of higher functions (Sittig 1985). Symptoms include a numbness and tingling sensation around the mouth, lips, and extremities; a clumsy stumbling gait; difficulty in swallowing and articulating words; a generalized sensation of weakness, fatigue and inability to concentrate; vision and hearing loss; spasticity and tremor; coma; and death (Amdur et al. 1991). Oral ingestion of organic mercury can cause damage to the central nervous system in both fetuses and children (ATSDR 1992b).

The risk evaluation of mercury is based on EPA-established toxicity values obtained from the EPA (1996). The ingestion RfD for mercury is 1.0E-04 mg/m^3 and is based on methyl mercury (organic mercury). An uncertainty factor of 3 is applied for variability in the human population, in particular the variation in the biological half-life of mercury and the variation that occurs in the hair:blood ratio for Hg. In addition, a factor of 3 is applied for lack of a two-generation reproductive study and lack of data for the effect of exposure duration on sequelae of the developmental neurotoxicity effects. The overall uncertainty factor is 10. Confidence in the RfD is medium, based on an evaluation of database and studies in the database. The inhalation RfC for mercury is 3.0E-04 mg/m^3 and is based on elemental mercury. The uncertainty of 30 is based on a factor of 10 used for the protection of sensitive human subpopulations together with the use of a LOAEL, and a factor of 3 used for the lack of database, particularly developmental and reproductive studies. Confidence in the RfC is medium, based on an evaluation of database and studies in the database. Mercury is not evaluated for carcinogenic effects because it is not classifiable as to human carcinogenicity.

5.9.3.2.15 Methylene Chloride—The critical effect of methylene chloride is liver toxicity (EPA 1996). The principal route of human exposure to methylene chloride is inhalation. Evaluation of pulmonary uptake in humans indicated that 70 to 75% of inhaled methylene chloride vapor is absorbed. Similar to other lipophilic organic vapors, methylene chloride absorption appears to be influenced by factors other than the vapor concentration. Increased physical activity and higher body fat increases the amount of methylene absorbed by the body (ATSDR 1993c).

Effects from inhalation of methylene chloride include headache, giddiness, stupor, irritability, numbness, and tingling in the limbs. Irritation to the eyes and upper respiratory passages occurs at higher dosages. In severe cases, observers have noted toxic brain disease with hallucinations, effusion of fluid into the alveoli and interstitial spaces of the lungs, coma, and death. Cardiac arrhythmias have been produced in animals but have not been common in human experiences. Exposure to methylene chloride may cause elevated carboxyhemoglobin levels that may be significant in smokers, or workers with anemia or heart disease, and those exposed to carbon monoxide (Sittig 1985).

The central nervous system is affected adversely in humans and animals at exposure levels of 500 ppm or higher. Noted effects from these exposure levels were decreased visual and auditory functions; however, these effects were reversible once exposure ceased. Similarly, psychomotor performance (reaction time, hand precision, steadiness) was impaired and alterations in visual evoked response have been observed in humans exposed to higher levels of methylene chloride (ATSDR 1993c).

The EPA classifies methylene chloride as a B2 probable human carcinogen (i.e., sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) for both ingestion and inhalation (EPA 1996). The risk evaluation of carcinogenic effects for methylene chloride is based on established EPA values. The oral SF for methylene chloride obtained from IRIS is 7.5E-03 $(mg/kg\text{-}d)^{-1}$, and the inhalation unit risk is 4.7E-07 $(\mu g/m^3)^{-1}$. Although important uncertainties remain regarding the pharmacokinetics,, and mechanisms of carcinogenicity for methylene chloride, the confidence in the toxicity values is medium.

The evaluation of noncarcinogenic effects after ingestion of methylene chloride is based on an EPA established chronic RfD, 6.0E-02 mg/kg-d (EPA 1995); the inhalation RfC for methylene chloride is 3.0E+00 mg/m^3 (EPA 1995a). The uncertainty factor of 100 accounts for both the expected intra- and inter-species variability to the toxicity of this chemical in lieu of specific data. The overall confidence in the oral RfD is medium due to the fact that the data base is rated medium to low because of the limited number of studies.

5.9.3.2.16 Nickel—The critical effect of nickel is decreased body and organ weights (EPA 1996). Toxicity information is available for both soluble salts of nickel and for inhalation of nickel refinery dust. Because any nickel found at WAG 9 would not come from refining operations, the health affects associated with this form of nickel is not presented.

The critical effects from ingestion of soluble salts of nickel are decreased body weight and organ (i.e., heart and liver) weights (EPA 1996). Two other sensitive endpoints are death of newborns and dermatotoxicity.

Approximately 8 to 10% of women and 1 to 2% of men tested demonstrated a sensitivity to nickel as determined in a patch test. Initial sensitization to nickel is believed to result from dermal contact (EPA 1996). Therefore, women are the sensitive subpopulation. However, because of several problems with

human studies (e.g., placebo effects observed, small numbers of individuals), animal studies were used to derive the RfD.

Reproductive effects have been seen in animals (e.g., decreased litter size, weight of pups, and number of live births). Dermal effects have also been observed after ingestion of nickel (EPA 1996). Ingestion may cause an eruption or worsening of a skin rash. For both dermal and reproductive effects a dose-response relationship is difficult to determine.

Nickel is considered to be an essential metal, and deficiency causes retarded body growth and anemia secondary to impaired absorption of iron from the gastrointestinal (GI) tract. Nickel is only sparsely absorbed from the GI tract and excretion in urine is nearly complete in 4 to 5 days (Amdur et al. 1991).

The oral RfD for nickel salts is 2E-02 mg/kg-d based on a NOEL (EPA 1996). An uncertainty factor of 300 is used to account for interspecies extrapolation, sensitive individuals, and inadequacies in the reproductive studies. There is low confidence in the study and medium confidence in the database and RfD. Low confidence in the study is based on 88% lethality in the control group and medium confidence in the database and RfD because of supporting evidence in a subchronic study with inadequacies in the reproduction data.

5.9.3.2.17 Nitrate—The critical effect of nitrate is early clinical signs of the presence of hemoglobin in an oxidized state in the blood (EPA 1996). Because nitrates can have adverse effects, sodium and potassium nitrate are evaluated for noncarcinogenic effects. The nitrate form of nitrogen is of concern because this ion is highly soluble in water, which enhances leaching, diffusion, and environmental mobility in soil and water.

The primary concern with nitrate in the environment is related to its conversion by biological systems to nitrite. Nitrite is formed from nitrate by certain microorganisms in the alimentary tract and in soil, water, and sewage (Amdur et al. 1991). Nitrate reduction to nitrite can occur under certain conditions in the stomach, as well as in the saliva. Nitrite acts in the blood to oxidize hemoglobin to methemoglobin, which cannot conduct oxygen to the tissues. This condition is known as methemoglobinemia and is caused in humans by high levels of nitrite or, indirectly, excessive levels of nitrate. Nitrate toxicity can result from ingestion of water and vegetables high in nitrates (EPA 1996). Infants (0 to 3 months) are more susceptible to nitrate toxicity than adults. This increased susceptibility has been attributed to high intake per unit weight, the presence of nitrate-reducing bacteria in the upper GI tract, the condition of the mucosa, and the greater ease of oxidation of fetal hemoglobin.

Other effects associated with ingestion of nitrates can include hypotension, relatively rapid heartbeat, respiratory depression (due to methemoglobinemia), headache, nausea, vomiting, and diarrhea. Convulsions have been reported following severe intoxication.

Little scientific basis exists to support conclusions concerning relationship between nitrate concentrations and carcinogenic potential (EPA 1996). Nitrates are not classified as carcinogens by the EPA. For this reason, nitrates are not evaluated for carcinogenic effects in this assessment.

The oral RfD for nitrate is 1.6E+00 mg/kg-d (EPA 1996). An uncertainty factor of 1 was employed because available data define the no-observed-adverse-effect level for the critical toxic effect in the most sensitive human subpopulation. Confidence in the RfD is high, based on an evaluation of the database and studies in the database.

5.9.3.2.18 Selenium—Selenium is not classifiable as to human carcinogenicity, except selenium sulfide is a B2 carcinogen (i.e., sufficient evidence in animals, but insufficient evidence in humans) (EPA 1994b). The critical effect from ingestion of selenium is clinical selenosis. In environmental exposures (i.e., low doses long period of time), chronic selenosis may occur that in humans is characterized by chronic dermatitis, fatigue, anorexia, gastroenteritis, liver degeneration, enlarged spleen, and increased concentration of selenium in hair and nails. Elemental selenium is poorly absorbed. Whether selenium is toxic or an essential metal depends on valence state when administered and incorporated into the biomolecules in the body (EPA 1994b).

The anticarcinogenic properties of selenium in humans are numerous and well documented (Amdur et al. 1991) whereas in animal studies, this effect is not consistently observed because of the different forms of selenium used and each form may have different bioavailabilities.

The recommended daily allowance (RDA) is 5 to 70 µg/day for North American females and males. This requirement increases during pregnancy, and children have higher intake requirements that vary with age. There have been some cases of teratologic effects in animals, and selenium has been shown to cross the placenta in animals. Other reproductive effects include decreased litter size and breeding events in animals (EPA 1994b)

The oral RfD for selenium is 5E-03 mg/kg-d and is based on a NOAEL from a human epidemiological study (EPA 1994b). An uncertainty factor of 3 is used because of human study at levels greater than the RDA with no selenosis occurring over a lifetime. This uncertainty factor accounts for sensitive individuals. Confidence in the critical study is medium with high confidence in data base and RfD. The medium confidence in the critical study because possible interactions with fluoride and protein status were not accounted for. One of the toxic forms of selenium binds with a protein (EPA 1994b).

5.9.3.2.19 Silver—Silver has been used in the treatment of syphilis and more recently it has been used as an astringent in topical preparations (EPA 1994b). The critical effects of silver ingestion is the permanent discoloring of skin to bluish-grey. This is from deposition of silver in the dermis and the silver induced production of melanin. Exposure of the affected area to sunlight will reduce the compound and cause increased pigmentation. No adverse health effects are associated with this discoloration (EPA 1994b). Irrespective of the pathway, very little silver is excreted from the body (Sittig 1985). Silver dust can cause irritation of the skin, burns of the conjunctiva, and blindness (Sittig 1985).

Silver is non-mutagenic and is not classifiable as to human carcinogenicity. The EPA-established toxicity value for ingestion is 5E-03 mg/kg-d (EPA 1994b). The uncertainty factor for this reference dose is 3 to account for minimal effects in a subpopulation which has exhibited an increased propensity for the development of argyria. There is medium confidence in the study because no information was presented for people who were given silver treatments but did not exhibit signs of skin discoloration. People in this study were being treated for syphilis and were of compromised health. There is a low confidence in the database because studies used to support the RfD were not controlled studies. There is low confidence in oral RfD because the RfD was obtained from a study where silver was administered intravenously and the dosage was converted to an oral RfD.

5.9.3.2.20 Zinc—Zinc is not classified as to human carcinogenicity (EPA 1994b). Zinc is an essential metal where deficiency results in severe health effects (Amdur et al. 1991). Excessive exposure is uncommon and requires high exposures because zinc does not accumulate in the body and approximately 70 enzymes require zinc. About 20 to 30% of the zinc ingested in absorbed in the body. The mechanism is

thought to be carrier-mediated process. Approximately 40% of the zinc absorbed is distributed to the liver, declining to 25% in 5 days. The greatest concentration of zinc in the body is in the prostate.

Penicillin has been shown to affect zinc homeostasis. Zinc deficiency may acerbate impaired copper nutrition and zinc interactions with cadmium and lead may modify the toxicity of these metals. Zinc supplements lower the HDL level in men and this can cause coronary artery disease; they have been shown to decrease the amount of iron in blood (EPA 1994b). No evidence of hematologic hepatic or renal toxicity have been observed when people ingest up to 12 g of elemental zinc over a 2 day period (Amdur et al. 1991). Recommend daily allowance range from 5 to 15 mg/d (EPA 1994b).

Zinc can be partially antagonistic to the reproductive effects of cadmium and lead. Zinc also interacts with proteins that could reduce the binding of drugs to these proteins and lessen the effect of the drug (Calabrese 1991).

Industrial exposure to inhalation of zinc fumes causes chills, fever, profuse sweating and weakness lasting from 24 to 48 hours and are most common on Mondays or after holidays (Amdur et al. 1991).

The oral RfD for soluble zinc salts is 3E-01 mg/kg-d (EPA 1994b). This RfD is based on an LOAEL that showed a 47% decrease in erthyrocyte superoxide dismutase concentration in adult females after a 10-week exposure. Erthyrocyte superoxide dismutase provides an indication of copper deficiency. This RfD allows for 79% of a 70-year lifetime (i.e., 55 years) that the dietary requirements will be met. There is medium confidence in the study, database, and RfD because there were only a few numbers of humans tested and the studies are all of short duration. The uncertainty factor is 3 because the most sensitive population (i.e., female adults) were tested.

5.10 Risk Characterization

5.10.1 Risk Characterization Methodology

Potential risks and hazards associated with the COPCs at the individual release sites are assessed for hypothetical current and future occupational and future residential receptors in the BRA. Nine potential exposure pathways were addressed, based on their applicability to each receptor exposure scenario and location:

1. Soil ingestion
2. Inhalation of fugitive dust
3. Inhalation of volatiles
4. External radiation exposure
5. Dermal absorption from soil
6. Groundwater ingestion (residential scenario only)
7. Ingestion of home grown produce (residential scenario only)

8. Dermal absorption of contaminants in groundwater (residential scenario only)

9. Inhalation of volatiles from indoor use of groundwater (residential scenario only).

The quantitative evaluation of the radionuclides and metals for each specific exposure pathway is dependent on the availability of toxicity values for exposure routes. Risks and hazard quotients were calculated only for those radionuclides and metals with established toxicity values. The carcinogenic risks were quantitatively evaluated in the BRA and compared to the NCP target risk range of 10^{-6} to 10^{-4}. Likewise, the noncarcinogenic effects were quantitatively evaluated based on the hazard quotient ratios relative to unity (e.g., 1). The methods used in the human health risk assessment follow EPA guidance (EPA 1989a). Thus, the risk values presented here are incremental individual lifetime cancer risks resulting from exposure to the radionuclides and metals at the individual sites.

Qualitative evaluations were provided in the individual risk assessments for metals where toxicity values were not available. In the qualitative risk assessments, the calculated concentration of the COPC is compared to occupational or other standards (e.g., maximum contaminant levels (MCLs) for drinking water).

5.10.1.1 Carcinogenic Effects Methodology. The potential for carcinogenic effects was evaluated by calculating the excess cancer risk from exposure to concentrations of radionuclides and metals based on hypothetical exposure scenarios. For carcinogens, risks represent the incremental probability of an individual developing cancer over a lifetime as a result of exposure to carcinogens.

The methodology for calculating cancer risks through the oral or inhalation exposure routes is to multiply the calculated carcinogenic intake (Section 5.3.3.1.1 through 5.3.3.4) by its appropriate toxicity value (Section 5.9) in accordance with the guidance (EPA 1989a):

$$R = I \times TV \tag{5-22}$$

where

R = cancer risk, expressed as a unitless probability

I = intake [(pCi), pCi-yr/g, mg/kg-d, μrem, $\mu g/m^3$]

TV = toxicity value [$(pCi)^{-1}$, $(pCi\text{-}yr/g)^{-1}$, $(\mu rem)^{-1}$, $(mg/kg\text{-}d)^{-1}$, or $(\mu g/m^3)^{-1}$].

Risk values for each evaluated exposure pathway are summed to obtain the total cancer risk for a given radionuclide or metal. The risks for the various radionuclides for each exposure pathway are also summed.

5.10.1.2 Noncarcinogenic Effects Methodology. The potential noncarcinogenic effects for the oral exposure routes were evaluated by comparing the estimated intake with the RfD. The resulting ratio is the hazard quotient and is defined as

$$HQ = \frac{I}{RfD} \tag{5-23}$$

where

HQ = hazard quotient

I = total intake (mg/kg-d)

RfD = reference dose (mg/kg-d).

For those metals that have a RfC for the inhalation exposure pathway (i.e., lead, manganese, and mercury), the hazard quotient is defined as

$$HQ = \frac{I}{RfC} \tag{5-24}$$

where

HQ = hazard quotient

I = intake (mg/m^3)

RfC = reference concentration (mg/m^3).

If the hazard quotient or hazard index (i.e., the sum of more than one hazard quotient is a hazard index) exceeds 1, there may be concern for the potential noncarcinogenic effects because the intake exceeds the RfD. If the hazard index is less than 1, the soil concentration of the metal is presumably below the threshold of potential noncarcinogenic effects, and no adverse health effects are expected from exposure to the metal.

5.10.2 BRA Risk Characterization Results

5.10.2.1 Hypothetical Current 0–25-year Occupational Exposure Scenario. The carcinogenic risks for all release sites at WAG 9 for the all exposure pathways and COPCs evaluated for the current occupational exposure scenario are presented in Table B-25. These risks are presented graphically in Figure 5-8. The risks for the Leach Pit and ANL-53 Riser Pits are not included since the depth to contamination is greater than 10 ft. The carcinogenic risks for ANL-53-North and ANL-53-South could not be calculated for the ingestion of soil pathway because for this pathway COCs do not have RfDs. The risks from the external radiation exposure pathway could not be calculated for ANL-01-Ditch B, ANL-53-North, ANL-53-South, and ANL-61A because radionuclides are not COCs. The risk from ingestion of soil pathway, at ANL-61A is 7E-05 and is within the NCP target risk range of 1E-4 to 1E-6. This PCB contaminated soil will be cleaned up in 1997 as a best management practice. For the inhalation of fugitive dust pathway, the calculated combined risk from all release sites is 2E-07 which is less then the lower limit of the NCP target risk range (1E-6). Also, the inhalation of volatiles pathway, is a combined risk from all release sites with a risk of 5E-09 that is less than the lower limit of the NCP target risk range (1E-6). The external radiation exposure pathway, three sites exceeded the upper limit of the NCP target risk range of (1E-4). These three sites are: ANL-01-IWP, ANL-09-Mound, and ANL-09-Canal with risks of 9E-04, 8E-04, and 5E-04, respectively with Ra-226 and Cs-137 being the only risk drivers. For the cumulative risk for the release sites, all release sites except ANL-01-IWP, ANL-09-Mound, and ANL-09-Canal were within the NCP target risk range. Again these sites risks were driven from the external

radiation exposure pathway from the Ra-226 and Cs-137 in ANL-01-IWP and only Cs-137 in ANL-09-Mound and ANL-09-Canal.

The hazard quotients for all exposure pathways and COPCs are presented in Table B-26. The hazard quotients for all exposure pathways for the current occupational exposure scenario are presented in Figure 5-9. The hazard quotients for ANL-09-Mound and ANL-61A for the ingestion of soil pathway could not be calculated because the mound only has radionuclide COCs which are carcinogens and ANL-61A has 4 ft of clean soil cover. All hazard quotients for each pathway, and hazard indexes for each release site are less than the lower limit of the NCP target risk range of 1.

5.10.2.2 Hypothetical Future 30-55-year Occupational Exposure Scenario. The carcinogenic risks for all release sites at WAG 9 for all exposure pathways and COPCs are presented in Table B-27. These risks are presented graphically in Figure 5-10. Similarly to the current 0-25 year occupational exposure scenario, not all risks could be calculated for the release sites. Risks were not calculated because either pathway was incomplete (ANL-53 Riser Pits) no carcinogenic COC present (ANL-53-North and ANL-53-South), or no carcinogenic RfDs (ANL-01-Ditch B, ANL-53-North, ANL-53-South, and ANL-61A). For the ingestion of soil pathway, the highest risks were at release sites ANL-01A and ANL-61A with risks of 1E-05 and 7E-05, respectively. These risks are within the NCP target risk range of 1E-04 to 1E-06. For the inhalation of fugitive dust and inhalation of volatiles pathways that are calculated cumulatively for all release sites showed risks of 2E-07 and 5E-09 respectively. These risks are less than those specified in the NCP target risk range of 1E-04 to 1-06. The highest risks for the external radiation exposure pathway were calculated for ANL-01-IWP, ANL-09-Mound, and ANL-09-Canal with risks of 5E-04, 4E-04, and 2E-04, respectively. These risks were exclusively from the levels of Ra-226 and Cs-137 in the ANL-01-IWP and Cs-137 in ANL-09-Mound and ANL-09-Canal. The risks exceed the upper limit of the NCP target risk range of 1E-04 to 1E-06. The cumulative risks for each release site were driven by the ingestion of soil and external radiation exposure pathways. The cumulative risks showed only ANL-01-IWP, ANL-09-Mound, and ANL-09-Canal with risks of 5E-04, 4E-04, and 2E-04, respectively, exceeding the NCP upper limit of 1E-04.

The hazard quotients for all exposure pathways and COPCs are presented in Table B-28. The hazard quotients for all exposure pathways for the current occupational exposure scenario are presented in Figure 5-11. The hazard quotients for ANL-09-Mound and ANL-61A for the ingestion of soil pathway could not be calculated because the mound only has radionuclide COCs which are carcinogens and ANL-61A has 4 ft of clean soil cover. All hazard quotients for each pathway, and hazard indexes for each release site are less than the lower limit of the NCP target risk range of 1.

5.10.2.3 Hypothetical Future 100-130-year Residential Exposure Scenario.

The carcinogenic risks for all release sites at WAG 9 for all pathways evaluated for the potential future residential exposure scenario (starting 100 years from now) are presented in Figure 5-12. These risks are presented in Table B-29. Only the risks that exceed the upper limit of the NCP target risk range will be addressed. The calculated risk from ingestion of soil pathway at ANL-61A is 6E-04. ANL-61A, with a calculated risk of 2E-04, is also the only site that exceeds the upper limit of the NCP target risk range for the ingestion of homegrown produce exposure pathway. This PCB-contaminated soil will be cleaned up in 1997 as a best management practice. For the inhalation of fugitive dust pathway, the calculated combined risk from all release sites is 5E-07 which is less then the lower limit of the NCP target risk range (1E-06). Also, the inhalation of volatiles pathway, is a combined risk from all release sites with a risk of 6E-08, which is less than the lower limit of the NCP target risk range (1E-06). For the external radiation exposure pathway, two sites exceeded the upper limit of the NCP target risk range of (1E-04). These sites are ANL-

01—IWP and ANL-09—Mound, with risks of 4E-04 and 1E-04, respectively, with Ra-226 and Cs-137 in the ANL-01—IWP and Cs-137 in the ANL-09—Mound being the risk drivers. For the cumulative risks from the ingestion of groundwater, dermal adsorption of groundwater, and inhalation of water vapors from indoor water use exposure pathways, the risks were calculated to be 3E-04, 9E-07, and 1E-03, respectively. The risk driver for the ingestion of groundwater and inhalation of water vapors from indoor water use is arsenic. Arsenic contributed all the risk (3E-04 and 1E-03) for these two pathways.

The hazard quotients for the future residential exposure scenario (starting 100 years from now) are presented in Figure 5-13. These hazard quotients are presented in Table B-30. The hazard quotient for residential ingestion of soil exposure pathway at the Industrial Waste Pond is 1 with arsenic contributing 0.3 and hexavalent chromium contributing 0.8. Three sites (ANL-01A-MCTBD, ANL-01-IWP, and ANL-01-Ditch B) had hazard indices of 1 for the ingestion of homegrown produce exposure pathway. Each of these sites had mercury and arsenic as the only two contributors to the hazard indices. For the cumulative pathways for inhalation of fugitive dust, inhalation of volatiles, ingestion of groundwater, and inhalation of water vapors from indoor water use, all hazard indices are less than 1 except for the ingestion of groundwater pathway. The hazard indices for ingestion of groundwater is 5, with arsenic and fluoride each contributing 1 and OCDD, 2,4,5-TP (Silvex), antimony, cadmium, selenium, and zinc each contributing slightly less than 1.

5.10.2.4 Hypothetical Future 1,000-1,030-yr Residential Exposure Scenario. The carcinogenic risks for all release sites at WAG 9 for all pathways evaluated for the potential future residential exposure scenario (starting 1,000 years from now) are presented in Figure 5-14. These carcinogenic risk calculations are also presented in Table B-31. Only the risks that exceed the upper limit of the NCP target risk range (1E-04) will be addressed. The calculated risk from ingestion of soil pathway at ANL-61A is 6E-04. ANL-61A, with a calculated risk of 2E-04, is also the only site that exceeds the upper limit of the NCP target risk range for the ingestion of homegrown produce exposure pathway. This PCB-contaminated soil will be cleaned up in 1997 as a best management practice. For the inhalation of fugitive dust pathway, the calculated combined risk from all release sites is 5E-07, which is less then the lower limit of the NCP target risk range (1E-06). Also, the inhalation of volatiles pathway is a combined risk from all release sites with a risk of 6E-08, which is less than the lower limit of the NCP target risk range (1E-06). For the external radiation exposure pathway, only ANL-01-IWP are exceeds the upper limit of the NCP target risk range of (1E-04) with a risk of 2E-04 from Ra-226. For the cumulative risks from the ingestion of groundwater, dermal adsorption of groundwater, and inhalation of water vapors from indoor water use exposure pathways, the risks were calculated to be 3E-04, 9E-07, and 1E-03, respectively. The risk driver for the ingestion of groundwater and inhalation of water vapors from indoor water use is arsenic. Arsenic contributed all the risk (3E-04 and 1E-03) for these two pathways.

The hazard quotients for the future residential exposure scenario (starting 1,000 years from now) are presented in Figure 5-15. The calculated hazard quotients are presented in Table B-32. The hazard quotient for residential ingestion of soil exposure pathway at the Industrial Waste Pond is 1 with arsenic contributing 0.3 and hexavalent chromium contributing 0.8. Three sites, ANL-01A-MCTBD, ANL-01-IWP, and ANL-01-Ditch B, had hazard indices of 1 for the ingestion of homegrown produce exposure pathway. Mercury and arsenic each contributed to the hazard indices for the homegrown produce exposer pathway for all three sites. For the cumulative pathways for inhalation of fugitive dust, inhalation of volatiles, ingestion of groundwater, and inhalation of water vapors from indoor water use, all hazard indices are less than 1 except for the ingestion of groundwater pathway. The hazard indices for ingestion of groundwater is 5, with arsenic and fluoride each contributing 1 and OCDD, 2,4,5-TP (Silvex), antimony, cadmium, selenium, and zinc each contributing slightly less than 1.

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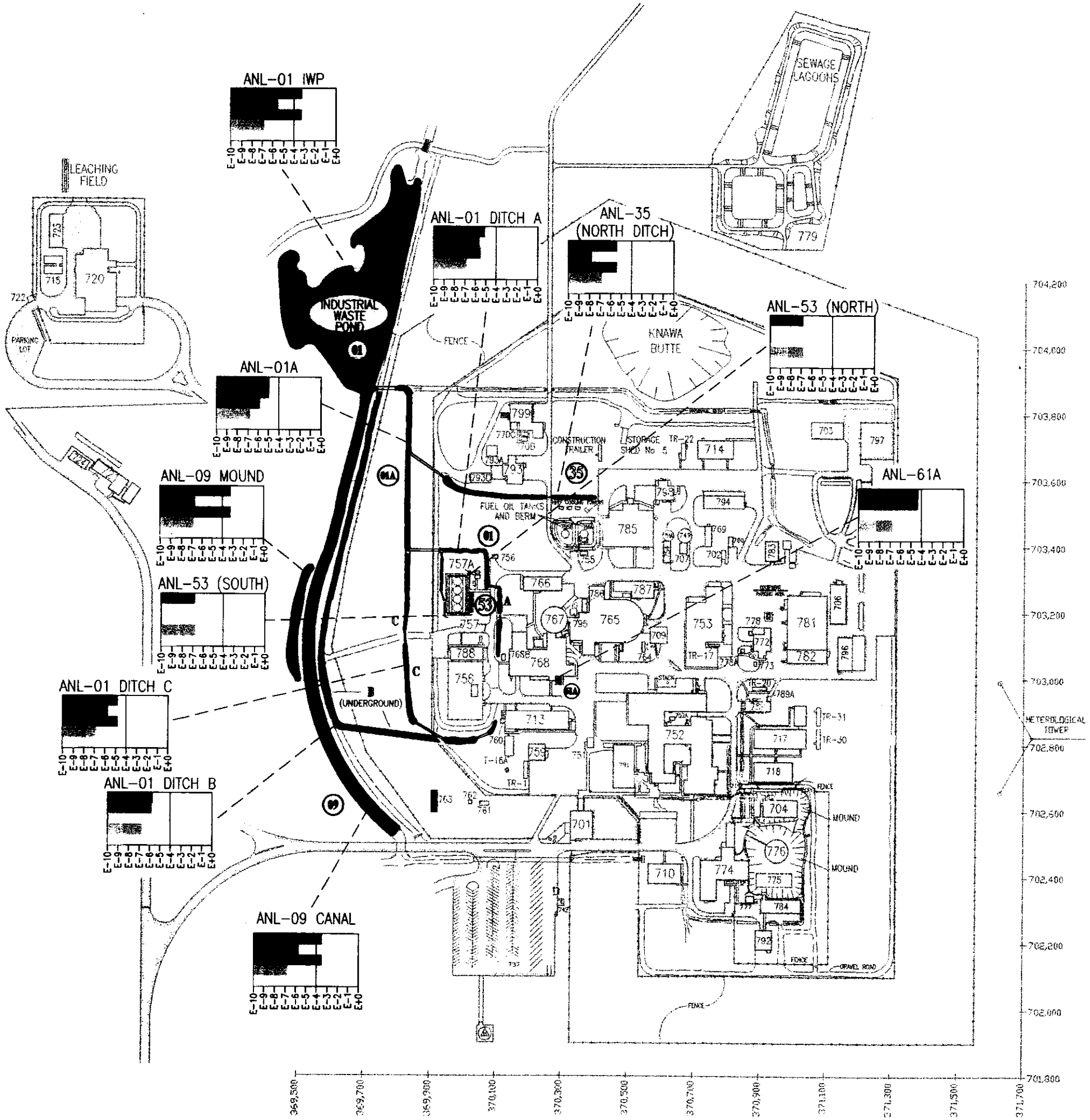
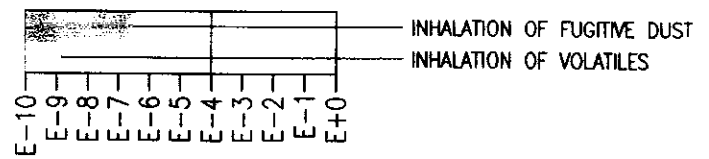


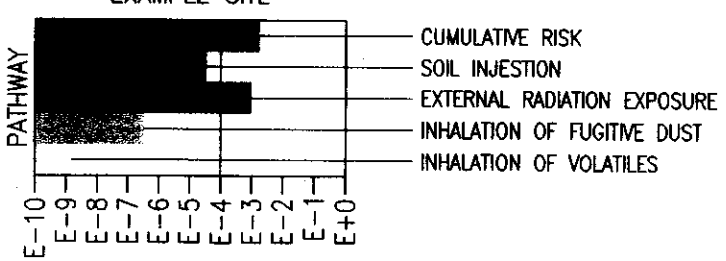
OPERABLE UNIT	SITE CODE	
9-01	ANL-W	(01A) EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	(02) EBR-II LEACH PIT
9-04	ANL-W	(01) INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	(01A) MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	(35) INTERCEPTOR CANAL
9-04	ANL-W	(36) INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	(53) RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG



EXAMPLE SITE



LEGEND

- ANL SITE
- WATER DEPOSITION SITE
- FENCE

N

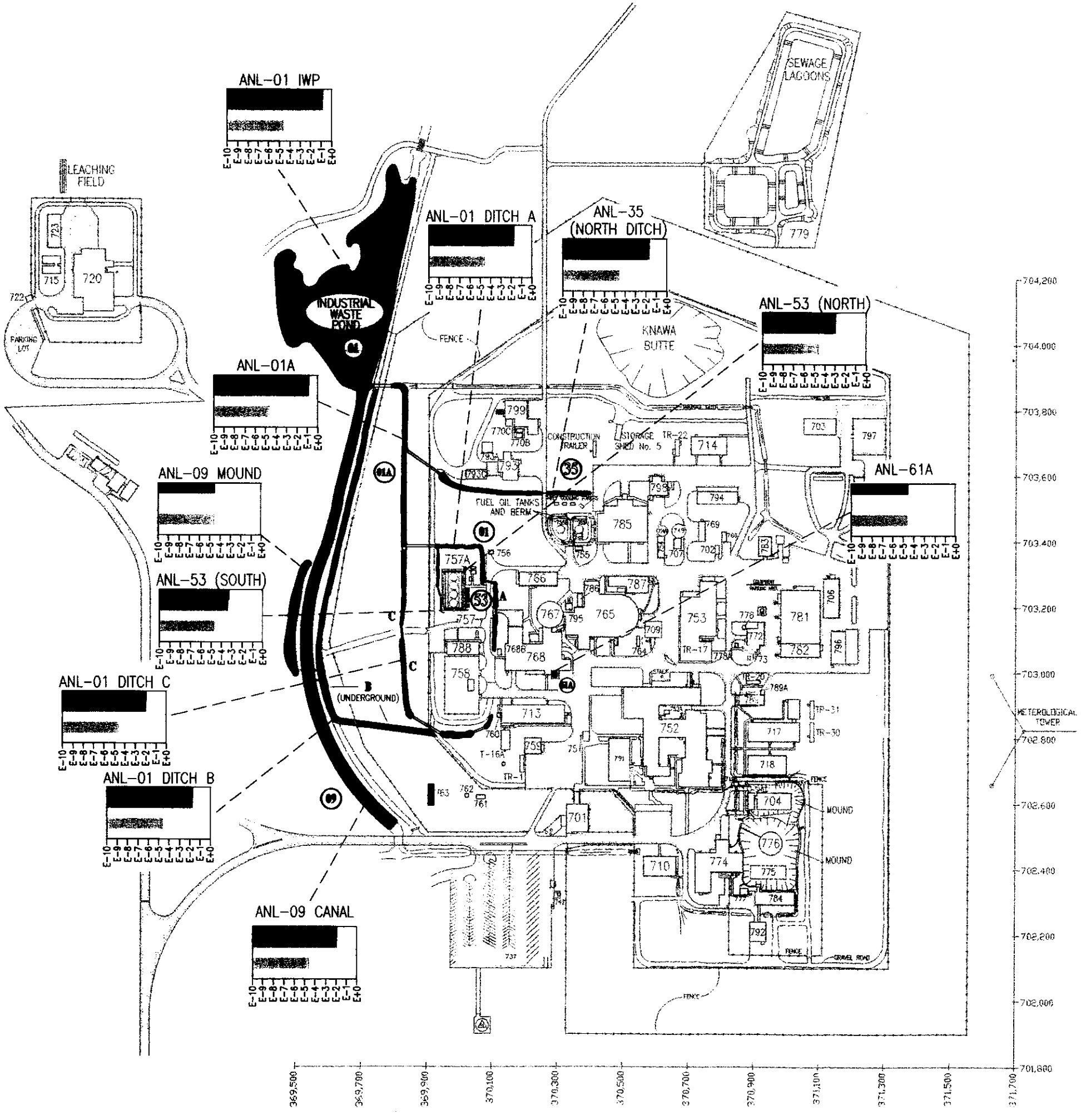
0' 100' 200' 300'

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-8 TOTAL RISK FOR WORKER AT 0 YEARS

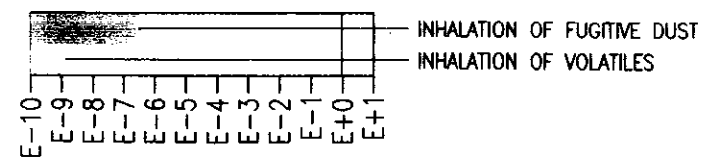
JUNE 12, 1997/ESWM
SITE-145.DWG (SITE-37)



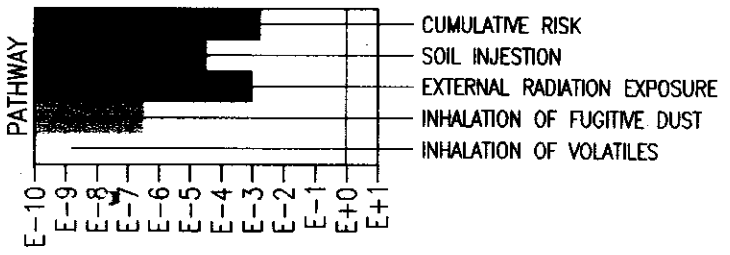
OPERABLE UNIT	SITE CODE	
9-01	ANL-W	(9A) EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	(9C) EBR-II LEACH PIT
9-04	ANL-W	(9D) INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	(9A) MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	(9F) INTERCEPTOR CANAL
9-04	ANL-W	(35) INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	(33) RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG



EXAMPLE SITE



LEGEND

ANL SITE

WATER DEPOSITION SITE

FENCE

N

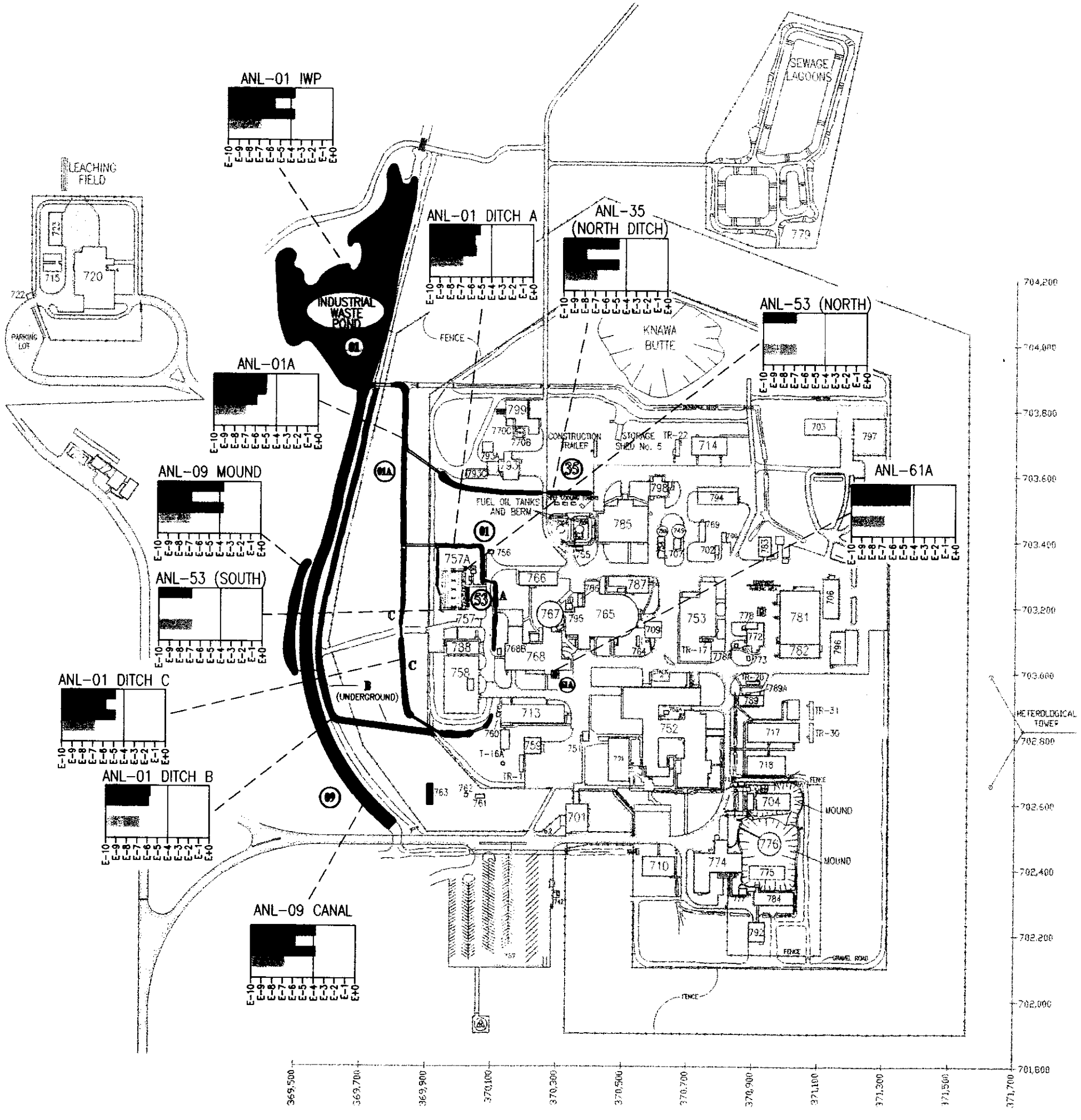
0' 100' 200' 300'

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-9 TOTAL HAZARD INDICES AT 0 YEARS

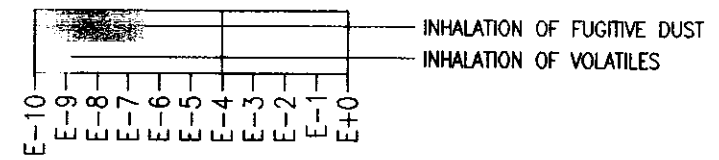
JUNE 12, 1997/ESWM
SITE-144.DWG (SITE-36)



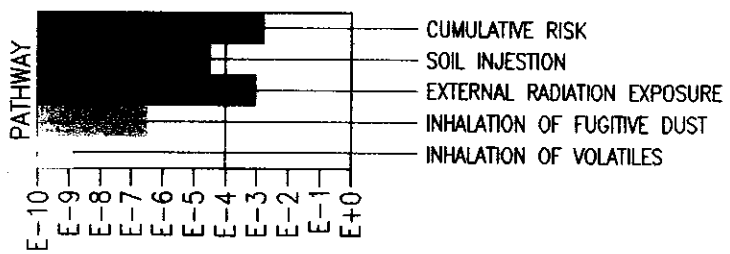
OPERABLE UNIT	SITE CODE		
9-01	ANL-W	24	EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	25	EBR-II LEACH PIT
9-04	ANL-W	01	INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	44	MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	35	INTERCEPTOR CANAL
9-04	ANL-W	55	INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	53	RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG



EXAMPLE SITE



LEGEND

ANL SITE

WATER DEPOSITION SITE

FENCE

N

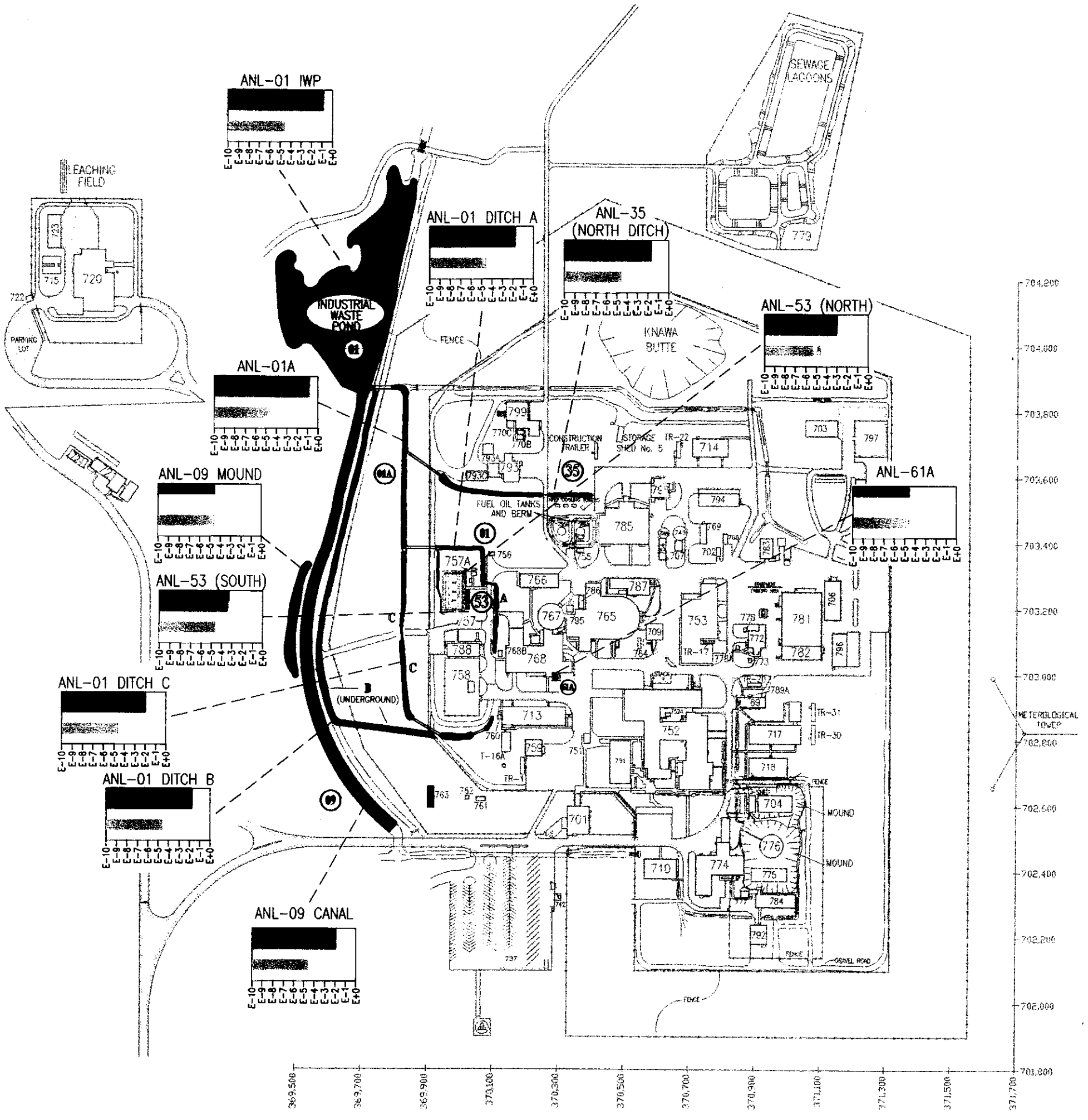
0' 100' 200' 300'

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-10 TOTAL RISK FOR WORKER AT 30 YEARS

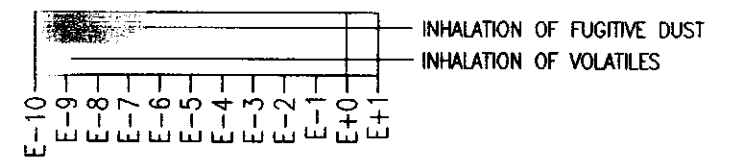
JUNE 12, 1997/ESWM
SITE-146.DWG (SITE-38)



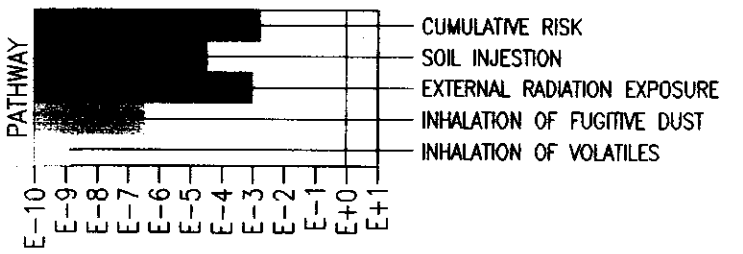
OPERABLE UNIT	SITE CODE	
9-01	ANL-W	(01) EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	(02) EBR-II LEACH PIT
9-04	ANL-W	(01) INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	(04) MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	(35) INTERCEPTOR CANAL
9-04	ANL-W	(53) INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	(53) RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG



EXAMPLE SITE



LEGEND

ANL SITE

WATER DEPOSITION SITE

FENCE

N

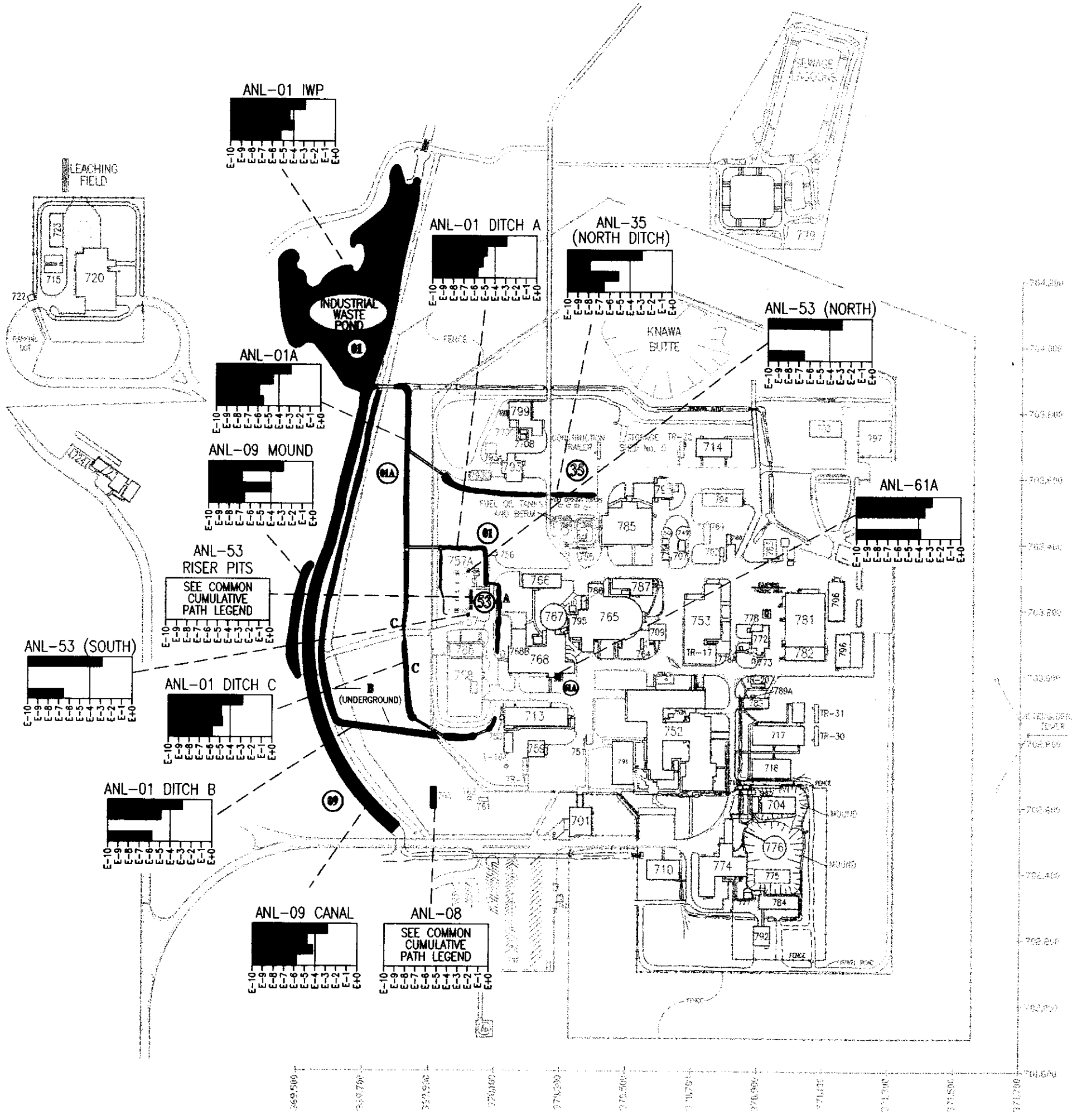
0' 100' 200' 300'

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-11 TOTAL HAZARD INDICES AT 30 YEARS

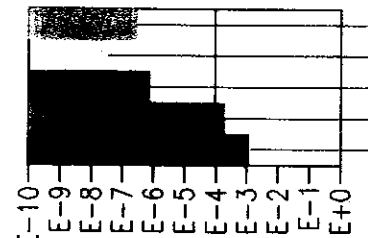
JUNE 12, 1997/ESWM
SITE-141.DWG (SITE-35)



OPERABLE UNIT	SITE CODE	
9-01	ANL-W	(9A) EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	(9C) EBR-II LEACH PIT
9-04	ANL-W	(01) INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	(9A) MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	(9) INTERCEPTOR CANAL
9-04	ANL-W	(35) INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	(53) RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG COMMON TO ALL RELEASE SITES



EXAMPLE SITE

LEGEND

- ANL SITE
- WATER DEPOSITION SITE
- FENCE

N

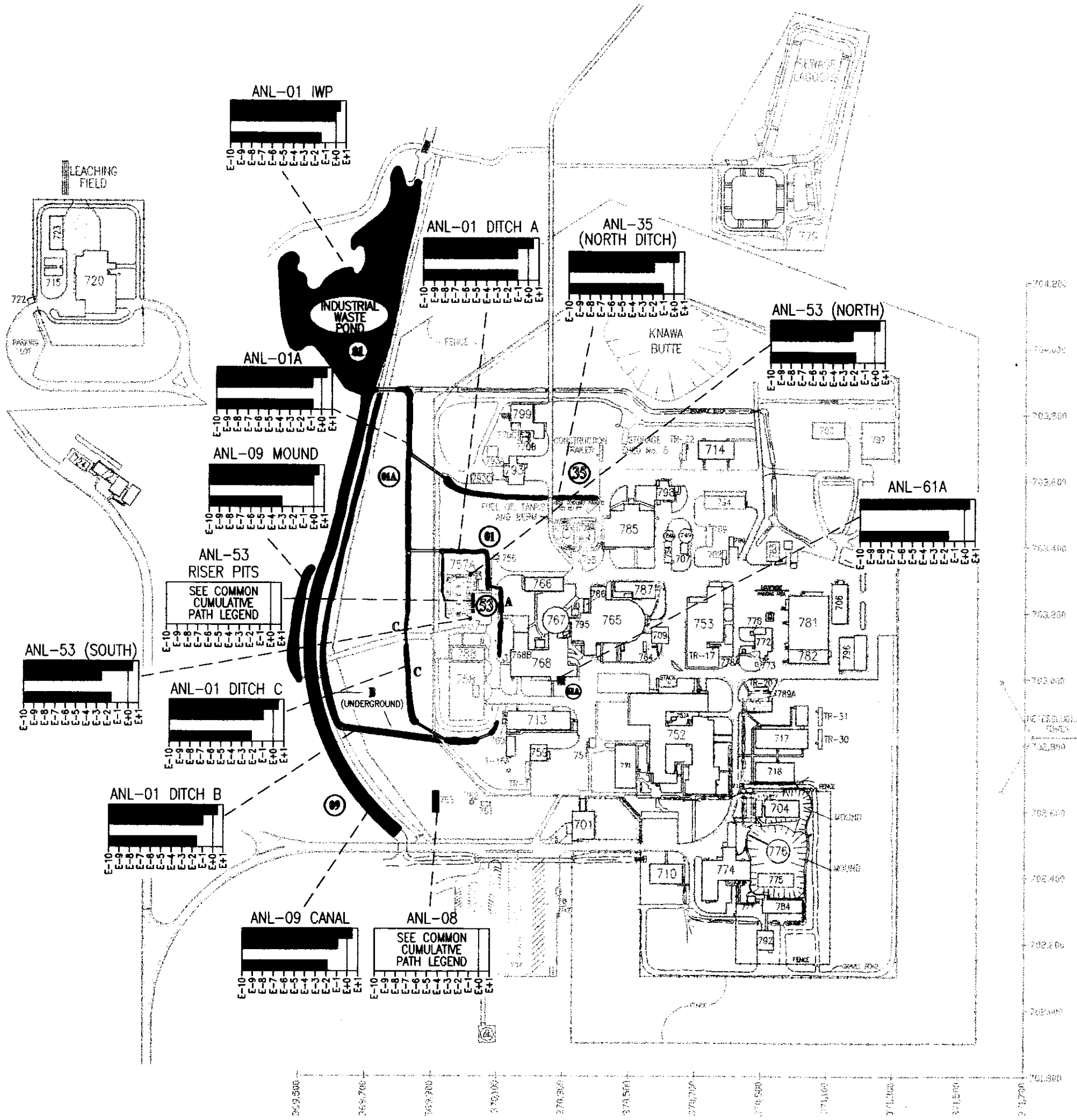
0' 100' 200' 300'

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-12 TOTAL RISK AT 100 YEARS

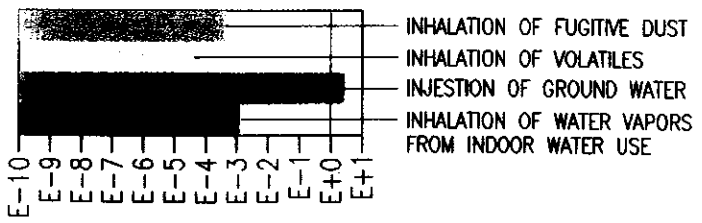
JUNE 12, 1997/ESWM
SITE-147.DWG (SITE-41)



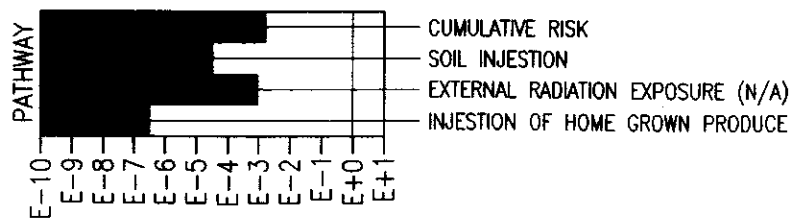
OPERABLE UNIT	SITE CODE	
9-01	ANL-W	(91A) EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	(90) EBR-II LEACH PIT
9-04	ANL-W	(01) INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	(44) MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	(35) INTERCEPTOR CANAL
9-04	ANL-W	(08) INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	(53) RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG COMMON TO ALL RELEASE SITES



EXAMPLE SITE



LEGEND

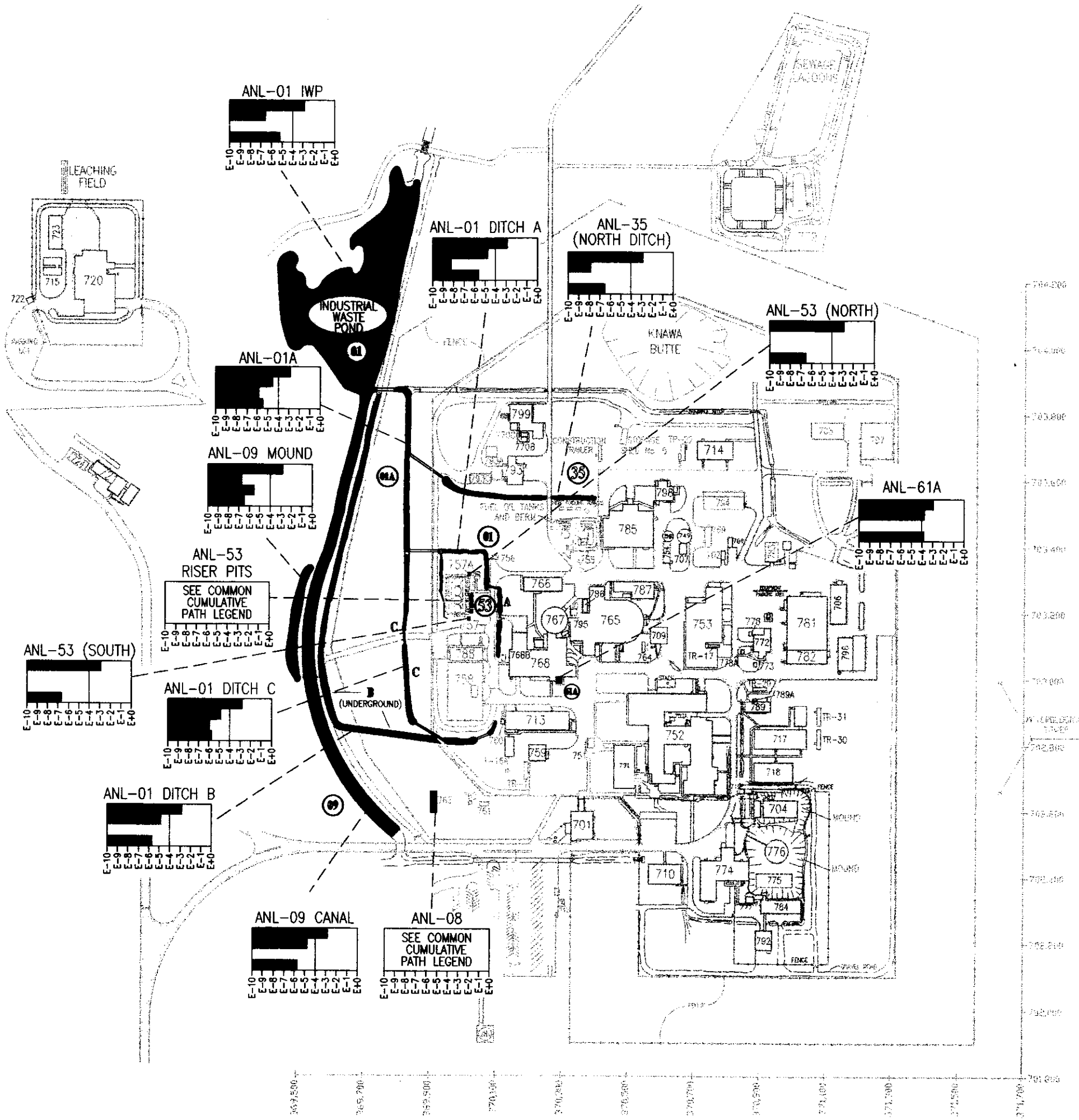
- ANL SITE
- WATER DEPOSITION SITE
- FENCE

0' 100' 200' 300'
SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-13 TOTAL HAZARD INDICES AT 100 YEARS

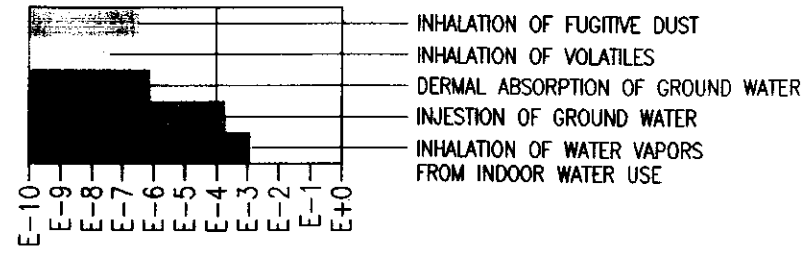
JUNE 12, 1997/ESWM
SITE-149.DWG (SITE-46)



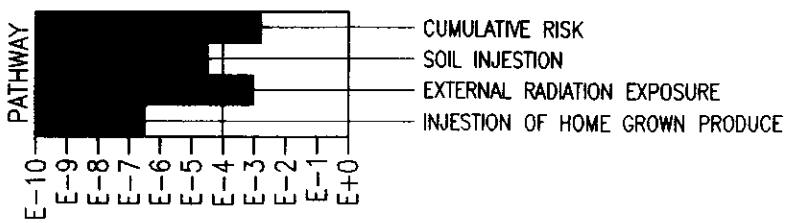
OPERABLE UNIT	SITE CODE	
9-01	ANL-W	(9A) EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	(9C) EBR-II LEACH PIT
9-04	ANL-W	(01) INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	(9A) MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	(09) INTERCEPTOR CANAL
9-04	ANL-W	(35) INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	(53) RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG COMMON TO ALL RELEASE SITES



EXAMPLE SITE



LEGEND

- ANL SITE
- WATER DEPOSITION SITE
- FENCE

N

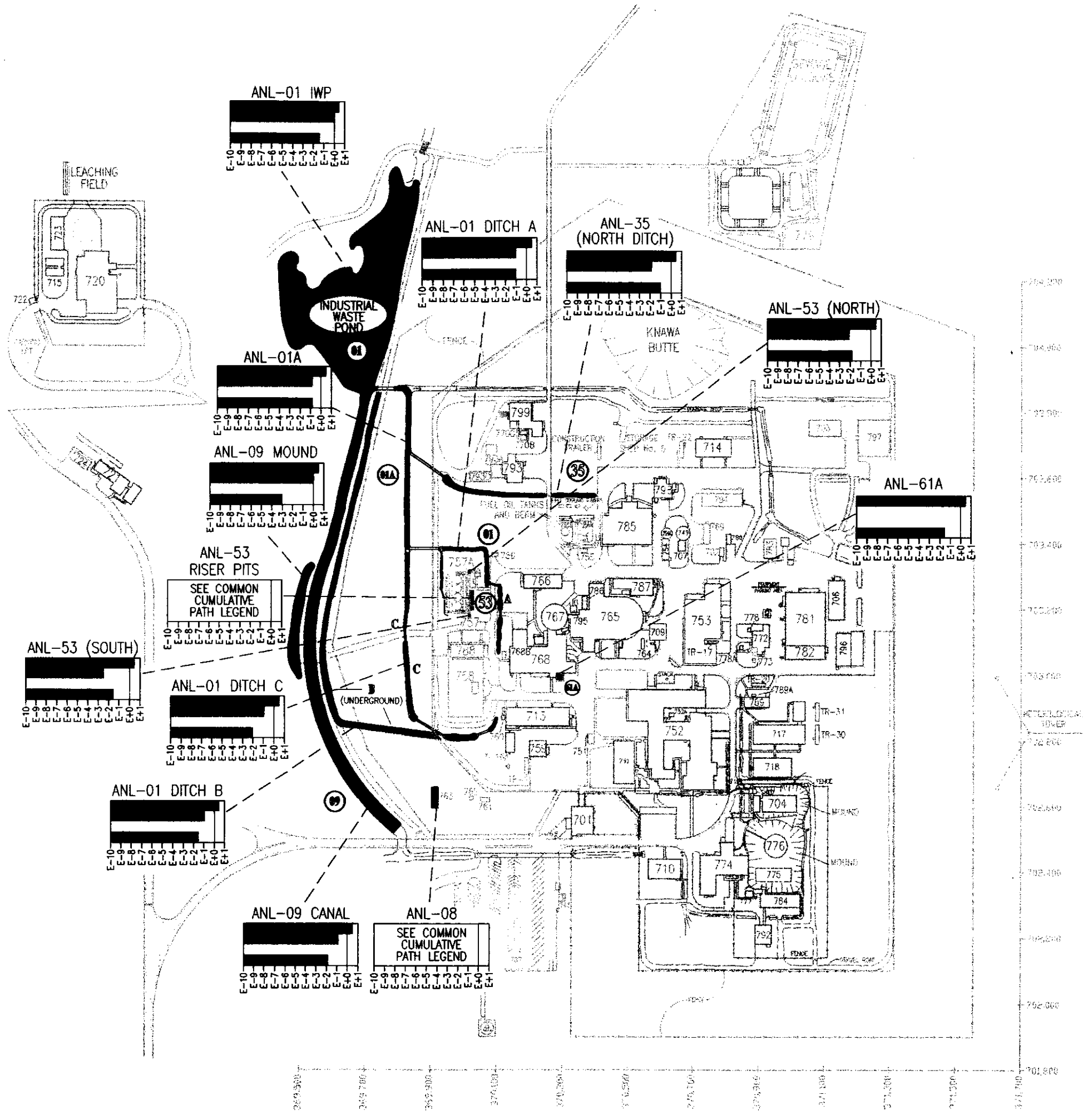
0' 100' 200' 300'

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-14 TOTAL RISK AT 1,000 YEARS

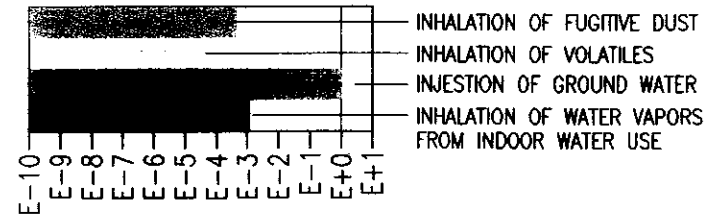
JUNE 12, 1997/ESWM
SITE-148.DWG (SITE-44)



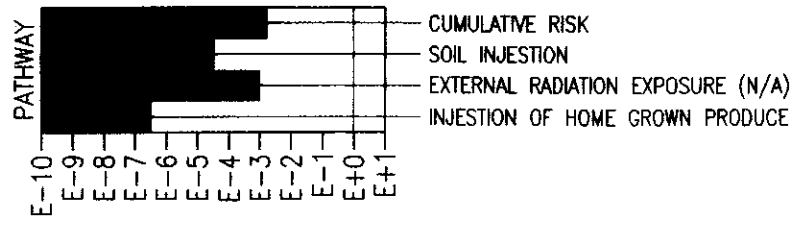
OPERABLE UNIT	SITE CODE		
9-01	ANL-W	21	EBR-II TRANSFORMER YARD ANNEX
9-02	ANL-W	36	EBR-II LEACH PIT
9-04	ANL-W	01	INDUSTRIAL WASTE POND AND THREE DITCHES (A, B, AND C)
9-04	ANL-W	24	MAIN COOLING TOWER BLOWDOWN DITCH (LDU)
9-04	ANL-W	07	INTERCEPTOR CANAL
9-04	ANL-W	35	INDUSTRIAL WASTE LIFT STATION DISCHARGE DITCH
9-04	ANL-W	53	RISER PITS

EXPLANATION

CUMULATIVE PATHWAY RISKS CALCULATED FOR THE WAG COMMON TO ALL RELEASE SITES



EXAMPLE SITE



LEGEND

- ANL SITE
- WATER DEPOSITION SITE
- FENCE

0' 100' 200' 300'

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES

FIGURE 5-15 TOTAL HAZARD INDICES AT 1,000 YEARS

JUNE 12, 1997/ESWM
SITE-150.DWG (SITE-47)

5.11 Risk Management

This section presents the human health risks from all the retained release sites at ANL-W and refines the list of sites that will be evaluated in the feasibility study. The refinement of the list of release sites will entail compiling summary tables of the risks, exposure scenarios and pathways, and screening the release sites that do not pose excessive risks. Section 5.11.1 compiles the summary tables of the ANL-W release sites and hazard indices. Section 5.11.2 presents the risk management screening of release sites. While Section 5.11.3 summarizes the ANL-W release sites that will be retained for evaluation in the feasibility study. The purpose of this section is to reduce the number of sites retained for evaluation in the feasibility study by eliminating those sites where the risks are less than the upper limit of the National Contingency Plan (1E-4), and to eliminate where either the pathway does not exist or the contaminant has been removed.

5.11.1 Risk Summary Tables

This section will present the carcinogenic risks and hazard indices for each of the release sites evaluated in the human health risk assessment. ANL-W has prepared Tables 5-34, 5-35, and 5-36 from the complete list of calculated risk values found in the Appendix B tables. This was done to aid the reader in evaluating which sites pose the greatest risks to the receptors. Each of these tables shows the release site, exposure scenario, exposure pathway, COC contributing to the risk, calculated risk or hazard quotient, and total exposure pathway excess cancer risk or hazard index. Table 5-34 shows only those sites that have exposure pathway excess cancer risks that are within the NCP risk range of 1E-4 to 1E-6. Table 5-35 shows only those sites that have exposure pathway excess cancer risks that are greater than the upper limit of the NCP (1E-4), while Table 5-36 shows only those sites that have an exposure pathway hazard index greater than 1.

5.11.2 Risk Management Screening

A human health baseline risk assessment was conducted for WAG 9. The assumptions used in conducting the baseline risk assessment tend to be very conservative. Some of the conservative assumptions used include; maximum contaminated soil volumes, eight hour a day exposure scenarios in the release sites over a 25 year or 30 year duration, uncertainty factors of up to 10,000 in development of reference doses and slope factors. The uncertainty factors are used to account for subpopulations that may be more sensitive to a contaminant or more sensitive because of their age (elderly or the young). Each of these assumptions tend to have an additive effect increasing the actual risks a receptor would have. Because under CERCLA we are obligated to protect all individuals not just those of average sensitivity, results in an overestimate of risks to those of average sensitivity. For example, if a receptor only works in a site for one hour a day for five years the calculated risk would be overestimated by 40 times [(8*25)/(1*5) = 40]. Worse yet, these conservative assumptions are cumulative in nature since they are multiplied with each other and result in an overestimate of risk for people with average sensitivity.

ANL-W has screened the calculated release sites using the following five step process:

- Eliminate sites with carcinogenic risks less than 1E-6,
- Eliminate sites with carcinogenic risks between 1E-4 and 1E-6,

Table 5-34. Contaminant risks greater than 1E-06 and less than 1E-04 for OU 9-04 exposure sites, scenarios, and pathways.

ANL-W Release Site	Exposure Scenario	Exposure Pathway	Contributing COC	Calculated Excess Cancer Risk	Exposure Pathway Excess Cancer Risk
ANL-01A-MCTBD	0-25- and 30-55-year Occupational	Ingestion of Soil	Arsenic	1E-05	1E-05
	0-25- and 30-55-year Occupational	External Radiation Exposure	U-238	2E-06	2E-06
	100- and 1,000-year Residential	Ingestion of Soil	Arsenic	5E-05	5E-05
	100- and 1,000-year Residential	External Radiation Exposure	U-238	4E-06	4E-06
	100- and 1,000-year Residential	Ingestion of Homegrown Produce	Arsenic	5E-06	5E-06
ANL-01-IWP	0-25- and 30-55-year Occupational	Ingestion of Soil	Arsenic	5E-06	5E-06
	0-25- Occupational	External Radiation Exposure	Co-60	6E-06	9E-04
	100- and 1,000-year Residential	Ingestion of Soil	Arsenic	7E-05	7E-05
	100- and 1,000-year Residential	Ingestion of Homegrown Produce	Arsenic	8E-06	8E-06
ANL-01-Ditch A	0-25- and 30-55-year Occupational	Ingestion of Soil	Arsenic	4E-06	4E-06
	0-25- and 30-55-year Occupational	External Radiation Exposure	U-238	5E-06	5E-06
	100- and 1,000-year Residential	Ingestion of Soil	Arsenic	3E-05	3E-05

Table 5-34. (Continued)

ANL-W Release Site	Exposure Scenario	Exposure Pathway	Contributing COC	Calculated Excess Cancer Risk	Exposure Pathway Excess Cancer Risk
	100- and 1,000-year Residential	External Radiation Exposure	U-238	9E-06	9E-06
	100- and 1,000-year Residential	Ingestion of Homegrown Produce	Arsenic	4E-06	4E-06
ANL-01-Ditch B	0-25- and 30-55-year Occupational	Ingestion of Soil	Arsenic	2E-06	2E-06
	100- and 1,000-year Residential	Ingestion of Soil	Arsenic	2E-05	2E-05
	100- and 1,000-year Residential	Ingestion of Homegrown Produce	Arsenic	3E-06	3E-06
ANL-01-Ditch C	0-25- and 30-55-year Occupational	Ingestion of Soil	Arsenic	2E-06	2E-06
	0-25- Occupational	External Radiation Exposure	Co-60	1E-06	2E-05
	0-25- and 30-55-year Occupational	External Radiation Exposure	U-238	2E-05	2E-05
	100- and 1,000-year Residential	Ingestion of Soil	Arsenic U-238	2E-05 2E-06	2E-05
	100- and 1,000-year Residential	External Radiation Exposure	U-238	3E-05	3E-05
	100- and 1,000-year Residential	Ingestion of Homegrown Produce	Arsenic	3E-06	3E-06
ANL-09-Canal	0-25- and 30-55-year Occupational	Ingestion of Soil	Arsenic	3E-06	3E-06

Table 5-34. (Continued)

ANL-W Release Site	Exposure Scenario	Exposure Pathway	Contributing COC	Calculated Excess Cancer Risk	Exposure Pathway Excess Cancer Risk
	0-25-year Occupational	External Radiation Exposure	Co-60	2E-06	5E-04
	100- and 1,000-year Residential	Ingestion of Soil	Arsenic	3E-05	3E-05
	100-year Residential	External Radiation Exposure	Cs-137	8E-05	8E-05
	100- and 1,000-year Residential	Ingestion of Homegrown Produce	Arsenic	3E-06	3E-06
ANL-09-Mound	0-25-year Occupational	External Radiation Exposure	Co-60	1E-05	
			U-238	2E-06	8E-04
	30-55-year Occupational	External Radiation Exposure	U-238	2E-06	4E-04
	100-year Residential	External Radiation Exposure	U-238	3E-06	1E-04
	1,000-year Residential	External Radiation Exposure	U-238	3E-06	3E-06
ANL-35	0-25-year Occupational	External Radiation Exposure	Co-60	2E-06	
			Cs-137	5E-05	
			U-238	2E-06	6E-05
	30-55-year Occupational	External Radiation Exposure	Cs-137	3E-05	
			U-238	2E-06	3E-05
	100-year Residential	External Radiation Exposure	U-238	3E-06	
			Cs-137	9E-06	1E-05
ANL-53 South	0-25- and 30-55-year Occupational	Ingestion of Soil	Arsenic	2E-06	2E-06
	100- and 1,000-year Residential	Ingestion of Soil	Arsenic	2E-05	2E-05
	100- and 1,000-year Residential	Ingestion of Homegrown Produce	Arsenic	3E-06	3E-06

Table 5-34. (Continued)

ANL-W Release Site	Exposure Scenario	Exposure Pathway	Contributing COC	Calculated Excess Cancer Risk	Exposure Pathway Excess Cancer Risk
ANL-61A	0-25- and 30-55-year Occupational	Ingestion of Soil	PCB's	7E-05	7E-05
All WAG 9 sites (Cumulative Pathway)	100- and 1,000 year Residential	Ingestion of Groundwater	Bis(2-Ethylhexyl) Phalate	4E-06	
			Methylene Chloride	7E-06	1E-06
	100- and 1,000 year Residential	Inhalation of water vapors from Indoor Water Use	Methylene Chloride	1E-06	1E-06
10-06 TREAT Windblown	30- year Residential	Ingestion of Homegrown Produce	Sr-90	2E-06	2E-06
10-06 Stockpile	100-year Residential	External exposure	Cs-137	1E-05	1E-05
All WAG 9 sites (Cumulative Pathway)	100- and 1,000 year Residential	Ingestion of Groundwater	Bis(2-Ethylhexyl) Phalate	4E-06	
			Methylene Chloride	7E-06	1E-06
	100- and 1,000 year Residential	Inhalation of water vapors from Indoor Water Use	Methylene Chloride	1E-06	1E-06

Table 5-35. Contaminant risks greater than 1E-04 for OU 9-04 exposure sites, scenarios, and pathways.

ANL-W Release Site	Exposure Scenario	Exposure Pathway	Contributing COC	Calculated Excess Cancer Risk	Exposure Pathway Excess Cancer Risk
ANL-01-IWP	0-25-year Occupational	External Radiation Exposure	Cs-137	8E-04	
			Ra-226	1E-04	9E-04
	30-55-year Occupational	External Radiation Exposure	Cs-137	4E-04	
			Ra-226	1E-04	5E-05
	100-year Residential	External Radiation Exposure	Cs-137	1E-04	
			Ra-226	2E-04	4E-04
	1,000-year Residential	External Radiation Exposure	Ra-226	2E-04	2E-04
ANL-09-Canal	0-25-year Occupational	External Radiation Exposure	Cs-137	5E-04	5E-04
	30-55-year Occupational	External Radiation Exposure	Cs-137	2E-04	2E-04
ANL-09-Mound	0-25-year Occupational	External Radiation Exposure	Cs-137	8E-04	8E-04
	30-55-year Occupational	External Radiation Exposure	Cs-137	4E-04	4E-04
	100-year Residential	External Radiation Exposure	Cs-137	1E-04	1E-04
ANL-61A	100-year Residential	Ingestion of Soil	PCBs	6E-04	6E-04
	1,000-year Residential	Ingestion of Soil	PCBs	6E-04	6E-04
	100-year Residential	Ingestion of Homegrown Produce	PCBs	2E-04	2E-04
	1,000-year Residential	Ingestion of Homegrown Produce	PCBs	2E-04	2E-04
All WAG 9 sites (Cum Pathway)	100- and 1,000-year Residential	Ingestion of Groundwater	Arsenic	3E-04	3E-04
	100- and 1,000-year Residential	Inhalation of vapors from indoor water use	Arsenic	1E-03	1E-03

Table 5-36. Contaminant hazard index greater than 1 for OU 9-04 exposure sites, scenarios, and pathways.

ANL-W Release Site	Exposure Scenario	Exposure Pathway	Contributing COC	Calculated Excess Hazard Quotient	Exposure Pathway Hazard Index
ANL-01-IWP	100- and 1,000 year Residential	Ingestion of Soil	Arsenic	0.3	
			Chromium (VI)	0.8	1
		Ingestion of Homegrown Produce	Zinc	0.4	
			Mercury	0.5	1
ANL-01-Ditch A	100- and 1,000 year Residential	Ingestion of Homegrown Produce	Zinc	0.1	
			Mercury	0.9	1
ANL-01-Ditch B	100- and 1,000 year Residential	Ingestion of Homegrown Produce	Zinc	0.8	
			Mercury	0.5	1
All WAG 9 sites (Cumulative Pathway)	100- and 1,000 year Residential	Ingestion of Groundwater	OCDD	3E-01	
			2,4,5-TP (silvex)	2E-01	
			Antimony	2E-01	
			Arsenic	1E+00	
			Cadmium	6E-01	
			Fluoride	1E+00	
			Selenium	2E-01	
			Zinc	2E-01	5

- Eliminate sites where the COC has been removed,

- Eliminate arsenic and silver as COC's based on localized background or natural accumulation, and

- Eliminate sites that have contaminant specific hazard quotients less than 1.

Each of the five risk management steps are described in more detail in the following sections.

5.11.2.1 Step 1, carcinogenic risks less than 1E-6,

All exposure pathways for the release sites with risks less than the lower limit of the NCP will be dropped from consideration in the feasibility study. The lower limit of the NCP is 1E-6 or a 1 in 1,000,000 chance of getting an excess case of cancer. These exposure pathways and sites are shown graphically in Figures 5-8 through 5-15 and have exposures less than the 1E-6 level.

5.11.2.2 Step 2, carcinogenic risks between 1E-4 and 1E-6

This step is based on the conservativeness of the risk assessment equations as explained above. ANL-W will drop from further consideration those pathways with total excess cancer risk within the risk range specified in the NCP (1E-4 to 1E-6). This means sites with calculated excess cancer risks for exposure pathways between 1 in 10,000 to 1 in 1,000,000 will be eliminated from further consideration in the feasibility study. This includes all release sites and pathways listed in Table 5-34. In addition all sites with a risk less than the lower limit of the NCP (1E-6) will also be dropped from further consideration in the feasibility study. Thus, all release sites with calculated excess cancer risks for exposure pathways less than 1 in 10,000 will be dropped from further consideration in the feasibility study. This step eliminates all release sites shown in Table 5-34 from consideration during the feasibility study.

5.11.2.3 Step 3, sites that either the COC or pathway has been removed

ANL-W currently has one release site that has risks greater than 1E-4 that is undergoing a cleanup in 1997 as a best management practice. This site is ANL-61A—PCB contaminated soil around an underground diesel tank. The underground diesel storage tank is being removed prior to the enforceable dates and the contaminated soil around the tank is being removed and disposed. The PCB contaminated soil is being removed to the specified risk based concentrations per exposure scenario and depths. Once the contaminated soil is removed the source of contamination in this unit is eliminated and the risks to receptors is also eliminated. Thus, this site will be eliminated from evaluation in the feasibility study. The best management practice removal will have to be completed prior to the signing of the record of decision for WAG 9. This step eliminates release site ANL-61A, as shown in Table 5-35, from evaluation in the feasibility study.

As stated previously, all exposure pathways were calculated using the standard default parameters for a baseline risk assessment. This means that for a risk to a receptor to occur there has to be a contaminant and an actual exposure pathway. If either of these are eliminated the risk is eliminated. To date, no exposure pathways have been eliminated at ANL-W.

5.11.2.4 Step 4, eliminate arsenic and silver as COC for concentrations below ANL-W levels or accumulation of natural sources

In the screening of COCs, ANL-W identified that the first step in retaining contaminants would be to compare the maximum sample concentration from a release site to that of the INEEL 95%/95% background concentration. After completing this, ANL-W noted that arsenic was retained as a COC in sites that did not receive arsenic from ANL-W operations. In addition, no background concentration for silver is established in the INEEL 95%/95% background document. This prompted ANL-W to review all previous sampling activities to evaluate the ANL-W site specific background for arsenic and silver. The evaluation of ANL-W site specific arsenic and silver levels is found in Appendix K of this report.

This review of two previous background sampling activities was conducted in areas that were undisturbed and upwind of potential sources. The data sets were combined and the 95% upper confidence level (UCL) was calculated. The local arsenic UCL of 11.16 mg/kg was then compared to the INEEL arsenic background concentration (7.4 mg/kg) and calculated UCL concentrations for the ANL-W release sites. The results of this comparison showed that of the four release sites that did not receive arsenic (ANL-01—Ditch A, ANL-01—Ditch B, ANL-01—Ditch C, and ANL-09—Interceptor Canal), all were below the ANL-W background concentration of 11.16 mg/kg. The four sites (ANL-01—Industrial Waste Pond, ANL-01A—Main Cooling Tower Blowdown Ditch, ANL-04—Sewage Lagoons, and ANL-53—Riser Pits) were above the 11.16 mg/kg local background concentration.

For silver, the local UCL of 6.12 mg/kg was calculated as shown in Appendix K. This localized concentration of silver was then compared to the INEEL background silver concentration of ND (non detected). The localized level is much greater than that of the INEEL background level and after using the localized silver concentration of 6.12 mg/kg, five sites (ANL-08—Leach Pit, ANL-01—Ditch A, ANL-01—Ditch B, ANL-01—Ditch C, and ANL-09—Interceptor Canal) would be screened because they have concentrations less than the localized level. Three sites (ANL-01—Industrial Waste Pond, ANL-01A—MCTBD, and ANL-35—North Ditch) would be retained because they have concentrations greater than the localized level. Ultimately, the use of the localized silver background does not effect the human health risk assessment because as shown on Tables 5-34, 5-35, and 5-36 the risks and hazard quotients associated with exposure to silver results in risks less than 1E-6 and 1, respectively. But, the localized silver concentration will effect the number of sites and potential cleanup levels for sites that have been retained because of risks for ecological receptors.

ANL-W will use the localized background concentration of arsenic and silver to screen the sites from being evaluated in the feasibility study. By doing this ANL-W has in effect determined that the release sites that have arsenic or silver at or below the localized background levels of 11.16 and 6.12 mg/kg, respectively, will not contribute any excess risks.

Table 5-34 shows the release sites, exposure scenarios, exposure pathways, and contributing COCs for the sites with risks between 1E-4 and 1E-6. These sites were eliminated from inclusion in the feasibility study in step 2. But, as shown in Table 5-35, arsenic exceeds the groundwater ingestion and inhalation of vapors from indoor water use for the future residential scenarios during the cumulative evaluation of all WAG 9 sites. Currently eight sites exceed the INEEL arsenic background concentration and contribute to this pathway. But, ANL-W has shown that only four of these sites actually exceed the local background concentration. ANL-W will screen the other four sites for arsenic (ANL-01—Ditch A, ANL-01—Ditch B, ANL-01—Ditch C, and ANL-09—Interceptor Canal) from the sites retained for the feasibility study for the cumulatively evaluated groundwater ingestion and inhalation of vapors from indoor water use pathways.

Of the four sites (ANL-01—Industrial Waste Pond, ANL-01A—Main Cooling Tower Blowdown Ditch, ANL-04—Sewage Lagoons, and ANL-53—Riser Pits) that exceeded the localized background level of arsenic, no known source of the arsenic exists. The one common item between all four sites is that they have received large quantities of effluent discharges. These effluent discharges consisted mainly of Snake River Plain Aquifer water and minor quantities of corrosion inhibitors used in the cooling tower and raw sewage in the Sewage Lagoons. The volume of water released to the Sewage Lagoons has averaged 4.15E+6 gallons per year (1994 and 1995) while the Industrial Waste Pond discharges from 1961 through 1994 averaged 30.87E+6 gallons (Table 3-19). During the operation of the EBR-II reactor from 1961 to 1995 the water discharged from the Cooling Tower was discharged to the Main Cooling Tower Blowdown Ditch which flowed into the Industrial Waste Pond. The water in the Industrial Waste Pond would then evaporate or infiltrate into the ground. While the actual volume of water discharged to the Cooling Tower Riser Pits is unknown, they were used as infiltration basins when water was removed from the large circulation pipes to prevent them from freezing.

In evaluating the elevated arsenic values in these four sites, ANL-W used the known volume of Snake River Plain Aquifer water discharged at each site along with the concentrations detected in each site to determine if the ratios of arsenic detected are proportional to the quantity of water they received. If they are, there is some process occurring that is precipitating the arsenic from the Snake River Plain Aquifer water. As shown in Table 3-24, the Snake River Plain Aquifer contains low concentrations of arsenic which have been detected in the wells near ANL-W. These concentrations ranged from 1.7 to 3.8 ug/L and were positively detected in nine of seventeen samples. Table 5-37 shows the UCL values, concentration above localized background levels, annual volume discharged, and the calculated increased concentrations of arsenic for the Industrial Waste Pond and Main Cooling Tower Blowdown Ditch. The Riser Pits are not included in Table 5-37 since the quantity of water discharged to this facility is not known. The increased arsenic concentrations are based on no know process at ANL-W that has contributed arsenic to the Sewage Lagoons. The concentration of arsenic above the localized background of 11.16 mg/kg was determined to be 5.11 mg/kg for the Sewage Lagoons. The ratio of the annual discharges to the Sewage Lagoons verses the Industrial Waste Pond was calculated to be 13.5% (4.15E+6/30.78E+6). Thus, the added concentration in the Industrial Waste Pond was calculated to be 37.85 mg/kg (5.11x100%/13.5%). The calculated increase in arsenic in the Industrial Waste Pond and the Main Cooling Tower Blowdown Ditch can then be compared to the actual concentrations above the localized background levels. Note that the calculated concentrations in the Industrial Waste Pond and the Main Cooling Tower Blowdown ditch are the same since the ditch was used to transport all the water to the Industrial Waste Pond. The calculated arsenic concentrations are all greater than the concentrations above the localized background levels of arsenic for these sites. This indicates that since no known contaminant source of arsenic at ANL-W and the calculated arsenic values are below the concentrations used in the risk assessment the arsenic is probably precipitating out of the groundwater in these ANL-W sites. Thus, ANL-W will eliminate the arsenic as a contaminant of concern because the arsenic appears to be buildup of a natural source and ANL-W has not added to these natural concentrations.

In addition, arsenic was not a COPC at WAG 9 until the INEEL background soil concentrations document (Rood, 1996) was published. Previous arsenic upper tolerance limit concentrations in soil from the Track 1 and Track 2 guidance documents ranged from 24.65 mg/kg at Central Facilities, 37.24 mg/kg at Power Burst Facility, 38.4 mg/kg at Test Area North, 88.92 mg/kg at Test Reactor Area with a background of 68.87 mg/kg These were used as screening values for assessments performed before the INEEL background document (Rood, 1996) was published. The true background concentration may be higher or lower than the current value, which is 7.4 mg/kg. Even though the arsenic concentrations at ANL-W and at other sites exceed the background level reported by the INEEL background document (Rood, 1996), the overall groundwater quality in the SRPA and the groundwater quality in the vicinity of

ANL-W has not been adversely affected, as reported in the USGS Report 91-4015 (Background concentrations of Chemical Constituents in the Vicinity of the INEL).

Table 5-37. Parameters for ANL-W sites with arsenic concentrations greater than background.

ANL-W Site	Upper Confidence Limit (mg/kg)	Concentration above localized background (mg/kg)	Annual Discharge Volume (gallons)	Calculated arsenic concentration added to sites (mg/kg)
Sewage Lagoons	16.27	5.11	4.15E+6	NA
Industrial Waste Pond	25	13.84	30.87E+6	37.85
Main Cooling Tower Blowdown Ditch	35.06	23.9	30.87E+6	37.85

NA-Not Applicable ratios of arsenic are based on the incremental increase in concentration based on the volume of water discharged to the Sewage Lagoons.

5.11.2.5 Step 5, hazard quotients less than 1

Hazard quotients were calculated for each COC for each exposure pathway. Hazard quotients are calculated for COC's that are known not to cause cancer A hazard quotient is the ratio of a single substance exposure level to a reference dose for the same time duration. The tolerance ability for humans varies and the reference dose is based on the most susceptible individuals and then multiplied by the uncertainty factors (up to 10,000). This produces a very conservative value for non-cancer causing COC's. The hazard quotients are then added together by exposure pathway to determine the hazard index. Because of the conservative nature of the calculations, ANL-W will eliminate sites with hazard quotients less than 1 from further analysis in the FS.

Table 5-36 shows the release site, exposure scenario, exposure pathway, COC, calculated COC hazard quotient, and pathway hazard index. As shown in this table, only two exposure pathways exceed the hazard index of 1. The hazard index for release site ANL-01—Industrial Waste Pond equaled 1 for the 100- and 1,000-year ingestion of soil. In the assessment of the cumulative pathway for all WAG 9 release sites, the hazard index of 5 was calculated for the ingestion of groundwater for the 100- and 1,000-year residential scenarios.

Site ANL-01—Industrial Waste Pond has only two contaminants that, when added together, have a hazard index equal to 1. This site and exposure pathways can be eliminated from further consideration since it equals the screening limit of 1.

For the ingestion of groundwater for future residents, risk is assessed cumulatively. That is all contaminants are modeled to the groundwater and the maximum concentration from the overlapping plumes is used in the risk assessment. The receptor well is located in the downgradient location that produces the

maximum contaminant concentrations. The conservativeness of these assumptions is that the probability of a well being placed in the spot that produces the maximum concentration of contaminants is very small, the concentrations of COC are always changing, and a receptor drinks the water for thirty years. Thus, ANL-W will evaluate each of the COC's that contribute to the hazard index. As shown in Table 5-36, the hazard index is 5 and only eight contaminants contribute to it. These contaminants consist of six inorganics and two organics. The inorganics include, antimony, arsenic, cadmium, fluoride, selenium, and zinc with hazard quotients of 0.2, 1, 0.6, 1, 0.2, and 0.2, respectively. The organics include OCDD with a hazard quotient of 0.3 and 2, 4, 5-TP (silvex) with a hazard quotient of 0.3. The two contaminants that contribute the highest hazard quotients are arsenic and fluoride.

5.11.3 Retained Sites

The goal of the risk management section was to review the human health risk assessment results and determine which sites will be retained for evaluation in the feasibility study. ANL-W made Tables 5-34, 5-35, and 5-36 which summarize the risks and hazard indices for the release sites and pathways at WAG 9. All sites and pathways on Table 5-34 were screened and eliminated from further consideration in the feasibility study based on the conservative nature of the calculations and they are within the NCP risk range of 1E-04 and 1E-06. All sites and pathways shown in Table 5-36 were screened and eliminated from inclusion in the feasibility study because of the conservative assumptions and none of the individual COC's had hazard quotients greater than 1. For Table 5-35, release site ANL-61A was screened from further evaluation in the feasibility study because the contaminated soil was removed during the summer of 1997 as part of the underground storage tank removal. The groundwater exposure pathway for the future residential scenario for the ingestion and inhalation of arsenic for the cumulative effects from four release sites (ANL-01—Industrial Waste Pond, ANL-01A—Main Cooling Tower Blowdown Ditch, ANL-04—Sewage Lagoons, and ANL-53—Riser Pits) is screened because the arsenic is attributed from a natural buildup of arsenic in precipitating out of the groundwater. Table 5-38 shows the release sites, exposure scenarios, exposure pathways, contributing COC, calculated COC risk, and exposure pathway excess cancer risk for the sites retained for evaluation in the feasibility study.

5.12 Uncertainty Analysis

The risk assessment results presented in this BRA are very dependent on the exposure assessment methodologies described in Section 5.3. These exposure assessments are based on EPA guidance, were refined over the period of several years by INEEL risk management and risk assessment professionals to provide realistic, and yet conservative, estimates of human health risks at INEEL. Nonetheless, if different risk assessment methods had been used, the BRA would likely have produced different risk assessment results. To ensure that the risk estimates are conservative, health protective assumptions that tend to bound the plausible upper limits of human health risks are used throughout the BRA. As a result, risk estimates that may be calculated by other risk assessment methods are likely to be significantly lower than the estimates presented in Section 5.10.

5.12.1 Sources of Uncertainty

The BRA results that are shown in Section 5.10, and evaluated in Section 5.11, are useful for evaluating which WAG 9 release sites require remediation, because the results are calculated in a consistent manner. This consistency allows for direct comparison of the risk assessment results for a given release site with the results for every other site included in the evaluation. Changes in a given assumption used in the evaluation would, in general, produce similar changes in the risk results for all of the release sites evaluated. As.

Table 5-38. Sites retained for evaluation in the feasibility study because of human health risks.

ANL-W Release Site	Exposure Scenario	Exposure Pathway	Contributing COC	Calculated Excess Cancer Risk	Exposure Pathway Excess Cancer Risk
ANL-01-IWP	0-25-year Occupational	External Radiation Exposure	Cs-137	8E-04	
			Ra-226	1E-04	9E-04
	30-55-year Occupational	External Radiation Exposure	Cs-137	4E-04	
			Ra-226	1E-04	5E-05
	100-year Residential	External Radiation Exposure	Cs-137	1E-04	
			Ra-226	2E-04	4E-04
	1,000-year Residential	External Radiation Exposure	Ra-226	2E-04	2E-04
ANL-09-Canal	0-25-year Occupational	External Radiation Exposure	Cs-137	5E-04	5E-04
	30-55-year Occupational	External Radiation Exposure	Cs-137	2E-04	2E-04
ANL-09-Mound	0-25-year Occupational	External Radiation Exposure	Cs-137	8E-04	8E-04
	30-55-year Occupational	External Radiation Exposure	Cs-137	4E-04	4E-04
	100-year Residential	External Radiation Exposure	Cs-137	1E-04	1E-04

described in the remainder of this section, the BRA results include inherent uncertainty, but despite this uncertainty, consistency of BRA process make the results useful for making remediation decisions

Uncertainty in this BRA is produced by uncertainty factors in the following stages of analysis:

1. Data collection and evaluation
2. Contaminant screening
3. Nature and extent of the contamination
4. Exposure assessment
5. Toxicity assessment
6. Risk characterization.

The following sections discuss each of these risk assessment stages in more detail, and Section 5.12.2 presents a discussion of risks due to potential future releases from co-located facilities at WAG 9.

5.12.1.1 Data Collection and Evaluation Uncertainties. Uncertainties associated with data collection and evaluation are produced by variability in observed concentrations due to sampling design and implementation, laboratory analysis methods, seasonality, contaminant level variation, and natural concentration variation. Optimizing the useability of sampling data involves quantifying these uncertainties.

The effect of uncertainty introduced from sample collection and analysis is reduced by using the upper 95% UCL for a log-normally distributed data set for the WAG 9 COPC. The resulting concentration estimates, used to estimate intakes, are an upper bound estimate of the concentrations observed at the retained sites. This approach is health-protective and accounts for the uncertainty introduced by sampling, analysis, seasonality, and natural variation.

A major assumption included in the BRA analysis is that all significant sources of contamination at WAG 9 have been identified and sampled. If a source of contamination has not been identified and subsequently sampled, the risks due to the contamination are not included in the BRA. In an effort to make sure that all sources have not fallen through the scope of this CERCLA BRA, all sites identified in the FFA/CO including those identified as No Action have been re-evaluated in the screening process (Section 3). In addition, co-located facilities at WAG 9 have been qualitatively assessed as potential sources (Section 5.12.2). The variabilities associated with the analysis of the samples collected by different sampling events for different years cannot be overlooked in the uncertainty section. Most of the OU 9-04 sites have been sampled more than once, and the associated analysis methods, detection limits, quality control, and data deliverable packages specified have changed. This uncertainty is easily shown by looking at the scatter plots of the data in Appendix A.

5.12.1.2 Contaminant Screening Uncertainties. The screening of the COPCs against the INEEL 95%/95% background values adds to the uncertainty of the COCs retained in the risk assessment. Since the INEEL is such a large facility covering more than 890 square miles, it is not fathomable that the background levels of analytes is homogeneous throughout the entire INEEL. Appendix K shows the ANL-W specific arsenic background of 11.16 mg/kg This is only slightly higher than the INEEL background

value of 7.4 mg/kg. The use of screening using the ANL-W background arsenic level eliminates all but two sites from exceeding the ANL-W background arsenic level.

5.12.1.3 Nature and Extent of Contamination Uncertainties. In the determination of each retained WAG 9 sites nature and extent of contamination. ANL-W used the maximum depth that the COPC was detected over the INEEL 95%/95% background value. This conservative assumption was used to calculate the nature and extent of the contamination to account for analyte variability between sampling locations. Ultimately, a site is never fully characterized until every gram of soil in that site has been analyzed. But with the use of statistics, the calculated 95% UCL of the mean will be used with some degree of confidence, eliminating the need to sample the entire site.

5.12.1.4 Exposure Assessment Uncertainties. Uncertainties associated with the exposure assessment are produced by characterizing transport, dispersion, and transformation of COPCs in the environment, establishing exposure settings, and deriving estimates of chronic intake. The initial characterization that defines the exposure setting for a site involves professional judgment and assumptions. Definition of the physical setting, population characteristics, and selection of the chemicals to be analyzed in the risk assessment are examples of areas for which a quantitative estimate of uncertainty cannot be achieved because of the inherent reliance on professional judgment. Assumptions and supporting rationale regarding these types of parameters, along with the potential impact on the uncertainty (i.e., over- or underestimation of uncertainty), are included in Table 5-39.

Another aspect of the risk assessment that tends to exaggerate risk results is the evaluation of contaminants with background concentrations that produce calculated risks in excess of 1E-06. Two examples of this type of contaminant are arsenic and beryllium. Both of these metals are commonly detected in INEEL soils at concentrations that are slightly higher than accepted background concentrations and both have very high toxicity constants. Since these contaminants have been detected at concentrations that are slightly above background levels and are associated with waste producing processes at WAG 9, they are included in the BRA. In this case, cleanup of the individual sites with arsenic contamination above the INEEL 95%/95% background values cannot be justified. Since the volume of arsenic contaminated soil at WAG 9 is deminimus in comparison to the total volume of soil at background for the INEEL that will cause a risk.

5.12.1.5 Toxicity Assessment. Several important measures of toxicity are needed in conducting an assessment of risks to human health. RfDs are applied to the oral and inhalation exposure to evaluate noncarcinogenic and developmental effects, and SFs are applied to the oral and inhalation exposures to carcinogens. RfDs are derived from NOAELs and the application of UFs and MFs. UFs are used to account for the variation in sensitivity of human subpopulations and the uncertainty inherent in extrapolation of the results of animal studies to humans, while MFs account for additional uncertainties in the studies used to derive the NOAEL or lowest-observed-adverse-effect-level (LOAEL). Uncertainty associated with SFs is accounted for by an assigned weight-of-evidence rating that reflects the likelihood that the toxicant is a human carcinogen. Weight-of-evidence classifications are tabulated and included while a discussion of the UFs and MFs used to derive RfDs are presented in Section 5.9.

5.12.1.6 Risk Characterization. The last step in the risk assessment is risk characterization. As discussed in Section 5.10, risk characterization is the process of integrating the results of the exposure and toxicity assessments. The uncertainties defined throughout the analysis process are combined and presented as part of the risk characterization to provide an understanding of the overall uncertainty in the estimate of risk. Table 5-34 presents this qualitative assessment of uncertainty.

The EPA recognizes that arsenic is double-entered to ensure that the risk assessor realizes that the non-carcinogenic concerns are significant for arsenic.

5.12.2 Co-Located Facilities/Structures Analysis

A co-located facility screening of all potential CERCLA hazardous substances release sites was conducted for WAG 9 in the *Final Operable Unit 9-04 Work Plan* (Lee et al. 1996). The approach to facility screening at WAG 9 is adapted from the approach developed by DOE-ID, IDHW/DEQ, and EPA WAG managers for WAG 3. The process was designed to ensure fulfillment of the requirements of CERCLA and evaluate the threat of release of hazardous substances to the environment. This process seeks to evaluate potential releases and to implement the D&D program under CERCLA, as specified by the DOE *Policy on Decommissioning Department of Energy Facilities Under CERCLA* May 22, 1995.

ANL-W used the five screening evaluation tables that were developed by WAG 3 at the Chemical Processing Plant. The ANL-W site plan, which lists the facility name and building numbers, is shown in Figure 5-16.

The WAG 9 site was subdivided into three separate evaluations. In the first evaluation, all above ground tanks and underground storage tanks (USTs) were screened. The second screening evaluated all the ANL-W facilities (buildings, towers, and parking lots). The third screening evaluated all tanks used to store potentially radioactive liquids. The completed screening tables are found in Appendix E for USTs, Appendix F for the facility screening, and Appendix J for the radiological liquid storage tanks of the *Final Operable Unit 9-04 Work Plan* (Lee et. al. 1996). The information in these appendices has not changed and thus the appendices will not be reissued in this RI/BRA report. ANL-W used four sources of information to complete the screening tables. The first was an ANL-W Surplus Inventory

Assessment-Phase II (copy provided to EPA and IDHW/DEQ WAG managers), the second was the ANL-W Termination Plan, the third was the 1995 annual SARA Title III report of chemicals in each facility, and the last was tours and interviews with ANL-W employees who operate these facilities.

The tanks at WAG 9 were evaluated using the screening tables. These tables are shown in Appendix E of the *Final Operable Unit 9-04 Work Plan* (Lee et. al. 1996). Of the 25 tanks that were evaluated, all 25 were eliminated as potential release sites. Typically, they were screened from further evaluation because they will be replaced prior to 1999 with aboveground tanks and/or the ANL-W spill prevention, control, and countermeasure program (Section 2.5 of ANL-W Environment Safety and Health Manual) provided a means of controlling a release. The screening process indicated that most WAG 9 tanks contained petroleum products. Most of the tanks in WAG 9 are used to supply diesel fuel to emergency generators for main facilities. These underground storage tanks will be removed or upgraded in accordance to UST regulations by December 22, 1998. Until then, the fuel volumes in the tanks are recorded weekly and compared with usage and fuelings. Typically the emergency generators used at ANL-W have small day tanks which are located in the building, thus eliminating the need to heat the tanks during the winter. These small day tanks with less than 25-gallon storage capacity were eliminated because they did not exceed the reportable quantity threshold under IDAPA 16.01.02.851.

Table 5-39. BRA human health assessment uncertainty factors.

Uncertainty Factor	Effect of Uncertainty	Comment
Source term assumptions	May overestimate risk	All contaminants are assumed to be completely available for transportation away from the source zone. In reality, some contaminants may be chemically or physically bound to the source zone and unavailable for transport.
Natural infiltration rate	May overestimate risk	A site specific conservative value was used for this parameter.
Moisture content	May overestimate or underestimate risk	Soil moisture contents vary seasonally in the upper vadose zone and may be subject to measurement error.
Water table fluctuations	May slightly overestimate or underestimate risk	The average value used is expected to be representative of the depth over the 30-year exposure period.
Mass of contaminants in soils estimated by assuming a uniform contamination concentration in the source zone.	May overestimate or underestimate risk	There is a possibility that most of the mass of a given contaminant at a given site may exist in a hotspot that was not detected by sampling. If this condition existed, the mass of the contaminant used in the analysis might be underestimated. However, 95% UCLs or maximum detected contamination were used for all mass calculations, and these concentrations are assumed to exist at every point in each waste site, so the mass of contaminants used in the analysis is probably overestimated.

Table 5-39. (continued).

Uncertainty Factor	Effect of Uncertainty	Comment
Plug flow assumption in groundwater transport	Could overestimate or underestimate risk	Plug flow models are conservative with respect to concentrations because dispersion is neglected, and mass fluxes from the source to the aquifer differ only by the time delay in the unsaturated zone (the magnitude of the flux remains unchanged). For nonradiological contaminants, the plug flow assumption is conservative because dispersion is not allowed to dilute the contaminant groundwater concentrations. For radionuclides, the plug flow assumption may or may not be conservative. Based on actual travel time, the radionuclide groundwater concentrations could be over or underestimated because a longer travel time allows for more decay. If the concentration decrease due to the travel time delay is larger than the neglected dilution due to dispersion, the model will not be conservative.
Chemical form assumptions	Could overestimate or underestimate risk	In general, the methods and inputs used in contaminant migration calculations, including assumptions made regarding chemical forms of contaminants were chosen in order to err on the protective side. All contaminant concentration and mass are assumed available for transport. This assumption results in a probable overestimate of risk.
Exposure scenario assumptions	May overestimate risk	The likelihood of future scenarios has been qualitatively evaluated as follows: - resident—improbable in ANL-W's opinion -industrial—credible in ANL-W's opinion The likelihood of future on-site residential development is small. If future residential use of this site does not occur, then the risk estimates calculated for future on-site residents are likely to overestimate the true risk associated with future use of this site.

Table 5-39. (continued).

Uncertainty Factor	Effect of Uncertainty	Comment
Exposure parameter assumptions	May overestimate risk	Assumptions regarding media intake, population characteristics, and exposure patterns may not characterize actual site exposures. ANL-W has developed more realistic site exposures and if the BRA indicates risks greater than the NCP range the ANL-W site specific exposure parameters will be used to quantify the risks.
Receptor locations	May overestimate risk	Groundwater ingestion risks are calculated for a point at a will being drilled in the maximum contaminate plume that is made from overlapping the individual contaminant plumes. The groundwater risk at this point is assumed to be the risk from groundwater ingestion at every point within the WAG 9 boundaries.
The entire inventory of each contaminant is assumed to be available for transport along each pathway	May overestimate risk	In reality, only a portion of each contaminant's inventory will be transported by each pathway.
Exposure duration	May overestimated	The assumption that an individual will work or reside at WAG 9 for 25 or 30 years is conservative. Short-term exposures involve comparison to subchronic toxicity values, which are generally less restrictive than chronic values.
Noncontaminant-specific constants (not dependent on contaminant properties)	May overestimate risk	Conservative or upper bound values were used for all parameters incorporated into intake calculations.
Exclusion of some hypothetical pathways from the exposure scenarios	May underestimate risk	Exposure pathways are considered for each scenario and eliminated only if the pathway is either incomplete or negligible compared to other evaluated pathways.
Model does not consider biotic decay	May overestimate risk	Biotic decay would tend to reduce contamination over time.

Table 5-37. (continued).

Uncertainty Factor	Effect of Uncertainty	Comment
Occupational intake value for inhalation is conservative	Slightly overestimates risk	Standard exposure factors for inhalation have the same value for occupational as for residential scenarios although occupational workers would not be on-site all day.
Use of cancer slope factors	May overestimate risk	Slope factors are associated with upper 95th percentile confidence limits for nonradioactive carcinogens only. They are considered unlikely to underestimate true risk.
Toxicity values derived primarily from animal studies	May overestimate or underestimate risk	Extrapolation from animal to humans may induce error due to differences in absorption, pharmacokinetics, target organs, enzymes, and population variability.
Toxicity values derived primarily from high doses, most exposures are at low doses	May overestimate or underestimate risk	Assumes linearity at low does. Tend to have conservative exposure assumptions.
Toxicity values and classification of carcinogens	May overestimate or underestimate risk	Not all values represent the same degree of certainty. All are subject to change as new evidence becomes available.
Lack of slope factors	May underestimate risk	COPCs without slope factors, may or may not be carcinogenic through the oral pathway.
Lack of RfDs	May underestimate risk	COPCs without RfDs may or may not have noncarcinogenic adverse effects.
Risk/HQs summed across pathways	May overestimate risk	Not all of the COPC inventory will be available for exposure through all applicable exposure pathways.

BUILDINGS

701	SECURITY GATE HOUSE
702	PLANT SERVICES EQUIPMENT STORAGE BUILDING
703	SODIUM STORAGE BUILDING
703A	SODIUM STORAGE FIRE EXTINGUISHER BUILDING
704	FUEL MANUFACTURING FACILITY (FMF)
704A	FMF COMPRESSOR BUILDING
705	FIRING RANGE
706	CONSTRUCTION MANAGEMENT/SPARE PARTS STORAGE BUILDING
707	FIRE PUMP HOUSE
708	SAFETY EQUIPMENT BUILDING
710	ENGINEERING OFFICE BUILDING
711	2000 KVA M/W SUBSTATION
713	MODULAR OFFICE BUILDING
714	MODULAR OFFICE BUILDING
716	MODULAR OFFICE BUILDING
718	DOE AREA GROUP—WEST
717	TECHNICAL CONSOLIDATION BUILDING
718	PROJECT BUILDING
719	SECURITY INSPECTOR POST BUILDING
720	TREAT REACTOR BUILDING
720A	TREAT SEPTIC TANK & LEACHING FIELD
721	TREAT OFFICE BUILDING
721B	TREAT OFFICE BUILDING SEPTIC TANK & LEACHING FIELD
722	TREAT GUARDHOUSE
723	TREAT WAREHOUSE
724	TREAT CONTROL BUILDING
737	PARKING LOT
742	GASOLINE/DIESEL DISPENSARY
748	400,000 GAL. WATER STORAGE TANK
750A	EBR-II EXPERIMENTAL EQUIPMENT BUILDING
750B	STORAGE SHED NUMBER 5
750C	INDUSTRIAL WASTE POND
751	OAB AND SAFETY STORAGE BLDG.
752	LABORATORY AND OFFICE BUILDING (L&O BUILDING)
752A	DIESEL GENERATOR BUILDING
753	PLANT SERVICES BUILDING
754	WELL PUMP HOUSE NO. 1
754A	200,000 GAL. WATER STORAGE TANK
755	FUEL OIL PUMP HOUSE
756A	100,000 GAL. FUEL OIL STORAGE TANK
756B	60,000 GAL. FUEL OIL STORAGE TANK
758	WELL PUMP HOUSE NO. 2
757	MAIN COOLING TOWER
757A	MAIN COOLING TOWER ACID SYSTEM BUILDING
758	ELECTRICAL SUBSTATION
759	FIRE HOUSE
760	SANITARY AND INDUSTRIAL WASTE PUMP HOUSE
761	REMOVED (FOUNDATION ONLY, BELOW GRADE)
762	REMOVED (FOUNDATION ONLY, BELOW GRADE)
763	LEACHING PIT (ABANDONED IN PLACE, BELOW GRADE)
764	SUSPECT STACK
765	FUEL CONDITIONING FACILITY (FCF)
766	SODIUM BOILER BUILDING
767	EBR-II REACTOR PLANT
768	EBR-II POWER PLANT
768B	WATER CHEMISTRY LABORATORY
768E	FLAMMABLE MATERIALS STORAGE
768F	EBR-II EMERGENCY BUILDING
769	DANGEROUS MATERIAL STORAGE BUILDING
770B	SODIUM COMPONENTS STORAGE BUILDING
770C	NUCLEAR CALIBRATION LABORATORY
771	RADIOACTIVE SCRAP AND WASTE FACILITY (RSWF)
772	EBR-II ENGINEERING LABORATORY
773	500 KW YARD SUBSTATION
774	ZPPR SUPPORT WING BUILDING
775	ZPPR WORKROOM—EQUIPMENT ROOM
776	ZPPR REACTOR CELL
777	ZPPR EQUIPMENT BUILDING
778	SANITARY SEWAGE LIFT STATION
778A	INDUSTRIAL WASTE LIFT STATION
779	SEWAGE LAGOONS
780	LAUNDRY SORTING BUILDING
781	MATERIAL HANDLING WAREHOUSE
782	MACHINE SHOP FACILITY
783	RIGGING TEST FACILITY
783A	FUEL DISPENSING STATION
784	ZPPR MATERIALS CONTROL BUILDING
785	HOT FUEL EXAMINATION FACILITY (HFEF)
785A	HFEF COOLING TOWERS
786	HFEF 480V. SUBSTATION
787	FUEL ASSEMBLY AND STORAGE BUILDING (FASB)
788	EBR-II MAINTENANCE SHOP
789	EBR-II ENGINEERING LABORATORY
789A	EQUIPMENT BUILDING
790	INTERIM CONTAMINATED EQUIPMENT STORAGE
791	INSTRUMENT AND MAINTENANCE FACILITY (IMF)
792	ZPPR MOCK-UP BUILDING
793	SODIUM COMPONENTS MAINTENANCE SHOP (SCMS)
793A	ALCOHOL STORAGE PAD
793B	SCMS ALCOHOL RECOVERY ANNEX
793C	CONTAMINATED STORAGE BUILDING
793D	SCMS SEPTIC TANK & LEACHING FIELD
794	CONTAMINATED EQUIPMENT STORAGE FACILITY
795	EBR-II COVER GAS CLEANUP SYSTEM BUILDING
796	METAL STOCK CONTROL BUILDING
797	RADIOACTIVE SODIUM STORAGE FACILITY (RSSF)
798	RADIOACTIVE LIQUID WASTE TREATMENT FACILITY (RLWTF)
799	SODIUM PROCESS FACILITY (SPF)
799A	CAUSTIC STORAGE TANK BUILDING

TRAILERS

TR-1	LIMTCO BUS DRIVERS TRAILER
TR-17	ELECTRICAL EQUIPMENT STORAGE
TR-20	EBR-II ENGINEERING LABORATORY
TR-22	GENERAL OFFICE SPACE
TR-30	GENERAL OFFICE SPACE
TR-31	GENERAL OFFICE SPACE

ADMINISTRATIVE BOUNDARY E 369,500.00'

ADMINISTRATIVE BOUNDARY N 701,500.00'

ARGONNE NATIONAL LABORATORY

ANL/W SITE PLOT PLAN

J. LEWIS — C6541 — 03/08/97

1 or 2

1 — IPE-750-1 — 40

The screening of all 90 facilities at WAG 9 was conducted using the WAG 3 screening tables (Appendix F) of the *Final Operable Unit 9-04 Work Plan* (Lee et al. 1996). Of the 90 facilities screened, only five were retained as potential release sites. These five facilities are 764-ANL-W 200 foot stack, 765-Fuel Conditioning Facility, 768-Power Plant, 787-Fuel Assembly and Storage Building, and 792-Zero Power Physics Reactor Mock-up Building. These were identified because they contained radioactive materials or could potentially release radioactive materials. A qualitative evaluation of these five facilities that failed the initial screening from the *Final Operable Unit 9-04 Work Plan* (Lee et al. 1996) is shown in Sections 5.11.2.1 through 5.11.2.5. Most of the facilities on the WAG 9 site are scheduled for decommissioning in 1998. All of the sodium currently stored at ANL-W will be treated at the Sodium Processing Facility under RCRA Part B permit application, and all fuel (depleted and nondepleted would be shipped off site for final disposal in accordance with the *Settlement Agreement* signed October 17, 1995, between the State of Idaho (Govenor Batt) and the Department of Energy.

ANL-W conducted the screening process on all 31 tanks at ANL-W that could contain or have contained potentially radioactive liquids. The screening tables of these 31 tanks is shown in Appendix J of the Final Operable Unit 9-04 Work Plan (Lee et al. 1996). All of these tanks are inside buildings in bermed areas and receive potentially radioactive liquids from decontamination showers, sink drains, janitorial mop water drains, and condensate from air conditioners. These liquids are hard piped directly to the storage tanks. When the volume in the tanks reaches approximately 50% the tanks are sampled for radionuclides, heavy metals, and organics. If the sample results show no hazardous substances the water is pumped to the sewage lagoons for disposal. If only radiological contamination is present, the wastes are pumped or trucked to the Radioactive Liquid Waste Treatment Facility. Other potentially radioactive liquid storage tanks receive their wastes from decontamination spray chambers and analytical testing fluids. These wastes typically contain radioactive liquids and are tested for hazardous constituents and pumped to the Radioactive Liquid Waste Treatment Facility for disposal (permitted RCRA TSD facility). All 31 tanks that currently contain or previously contained radioactive liquids were screened from the evaluation as shown in Tables 1 through 5 of Appendix J of the Final Operable Unit 9-04 Work Plan (Lee et l. 1996).

5.12.2.1 764- ANL-W 200 Foot Stack. The 200-foot main stack is located by the Fuel Conditioning Facility. The main stack is used to dispose of offgas from FCF and the EBR-II reactor building. The off-gas is continuously monitored for radionuclides; monitors are set to alarm at conservative setpoints if radionuclides are detected in the offgas from either FCF or the EBR-II reactor building. If an alarm condition exists, ANL-W has specific procedures in place to enable immediate response to minimize the release, keeping release levels well below allowable limits. These procedures are maintained in the FCF Emergency Plan and in the ANL-W Emergency Management Plan.

Any emissions from the stack are recorded as part of the annual air release report for the INEEL. Even though the stack is the point of release for any potential releases, the stack is eliminated from future evalation because it is not the source for CERCLA hazardous substances; FCF or the EBR-II reactor would be the source. The stack, upon cessation of operations in the FCF will be part of the decommissioning activities for FCF.

5.12.2.2 765 Fuel Conditioning Facility. The initial purpose of the FCF was the demonstration of the fuel fabrication cycle for the Integral Fast Reactor (IFR) Project. The IFR project has been terminated. FCF will be utilized in the future for the treatment of EBR-II spent fuel and for demonstrating DOE spent fuel treatment technologies. These demonstration technologies started operation on Friday June 7, 1996. Demonstration technologies are performed in hot cells within FCF. These hot cells have been designed to contain radioactivity thereby preventing release to the environment. Facility operations and emergency procedures currently implemented minimize the potential for release of

radioactivity to the environment. Ventilation systems are equipped with High Efficiency Particulate Air (HEPA) filters which are 99.97% efficient. Additionally, ventilation systems are designed and operated to provide a slight negative pressure, drawing the clean outside air to inside the more contaminated cells; air is then discharged through the HEPA filters. Monitors in the ventilation systems are set to alarm at conservative setponts if radionuclides are detected. These monitors are not used as mitigatory devices to prevent a release, but rather to provide early detection so that operators can correct abnormal conditions that could lead to exceeding levels allowed by the operating permit. Upon receipt of an alarm, personnel immediately respond. ANL-W has procedures in place which enable the operators to stop potential release activities after an alarm setpoint is encountered. These procedures are contained in the FCF Emergency Plan and Procedures and the sitewide ANL-W Emergency Management Plan. The site is not considered a potential release site under current operating conditions because the facility's operations, maintenance, and staffing by highly qualified personnel provide effective measures to prevent release of radionuclides. When the FCF's status changes, e.g. identified for decontamination and decommissioning, the facility will be evaluated in accordance with DOE Directive 5820.2A, Radioactive Waste Management, which requires that applicable regulatory requirements (RCRA, CERCLA, NEPA) be incorporated in the development of the decontamination and decommissioning program and project plans.

5.12.2.3 768-Power Plant. The EBR-II Power Plant houses the electrical power generation and reactor support systems for the EBR-II reactor. The power plant is currently in standby mode and has a sprinkler system in place for fire suppression. Chemicals are currently stored in appropriate cabinets located at various places in the building. Only a small number of chemicals are used for water chemistry and each is individually packaged. Some of the water chemistry chemicals have already been moved to other buildings for use. Current DOE long-range plans are to place the EBR-II reactor and support facilities in a radiologically and industrially safe shutdown condition pending decontamination and decommissioning. This site is removed from future CERCLA evaluation because this facility is already addressed in the EBR-II termination plan. Section 10.4 of the termination plan identifies that closure of plant systems and facilities will include the removal of hazardous items. Chemicals currently stored in appropriate cabinets located at various places in the building will be removed and disposed of in accordance with applicable regulatory requirements. Final decommissioning dates for this facility are unknown at this time.

5.12.2.4 787-Fuel Assembly and Storage Building. The Fuel Assembly and Storage Building (FASB) contains the Reactor Materials Lab, which consists of mechanical testing equipment and an electron microscope. The chemicals maintained in this facility are in small containers and are properly stored according to manufacturer specifications and the ANL-W Environmental Safety & Health Manual (4.1 G, Uniform Color Code; 4.1H, Housekeeping and Storage; 5.4, Employee Notification). Disposal of any unused products is in accordance with RCRA regulations. The facility currently stores radioactive materials. The building exhaust is continuously monitored for any radionuclide operations and emergency procedures currently implemented minimize the potential for release of radioactivity to the environment. Ventilation systems are equipped with High Efficiency Particulate Air (HEPA) filters which are 99.97% efficient. Additionally, ventilation systems are designed and operated to provide a slight negative pressure, drawing the clean outside air to more contaminated cells inside the building; air is then discharged through the HEPA filters. Monitors, for the ventilation systems are set to alarm if radionuclides are detected. These monitors are not used as mitigatory devices to prevent a release, but rather to provide early detection so that operators can correct abnormal conditions that could lead to exceeding levels allowed by the operating permit. Upon receipt of an alarm, personnel immediately respond. ANL-W has procedures in place which enable the operators to stop potential release activities after an alarm setpoint is encountered. These procedures are contained in the FCF Emergency Plan and Procedures and the sitewide ANL-W Emergency Management Plan. The site is not considered a potential release site under current operating conditions

because the facility's operations, maintenance, and staffing by highly qualified personnel provide effective measures to prevent release of radionuclides. When the FASB's status changes, e.g. identified for decontamination and decommissioning, the facility will be evaluated in accordance with DOE Directive 5820.2A, Radioactive Waste Management, which requires that applicable regulatory requirements (RCRA, CERCLA, NEPA) be incorporated in the development of the decontamination and decommissioning program and project plans.

5.12.2.5 792-Zero Power Physics Reactor Mock-Up Building. The ZPPR Mock-up Building is part of the ZPPR complex and provides a storage area for reactor experiment materials and a work area for assembling drawers of experiment materials for use in ZPPR. The building houses radioactive materials (uranium oxide) used for fuel assemblies. These radioactive materials are stored in appropriate containers within the facility. These radioactive materials are handled in accordance with strict facility specific procedures to ensure safety and nuclear material accountability. The site is not considered a potential release site under current operating conditions because the facility's operations, maintenance, and staffing by highly qualified personnel provide effective measures to prevent release of radionuclides. When the ZPPR's status changes, e.g. identified for decontamination and decommissioning, the facility will be evaluated in accordance with DOE Directive 5820.2A, Radioactive Waste Management, which requires that applicable regulatory requirements (RCRA, CERCLA, NEPA) be incorporated in the development of the decontamination and decommissioning program and project plans.

5.12.2.6 Summary of Risk From Co-Located Facilities/Structures. The facility screening for potential release sites at WAG 9 used the screening tables previously used at WAG 3. The WAG 9 site was divided into three separate studies: the screening of USTs, facilities, and potential radioactive liquid storage tanks. All of the 25 USTs and 31 potential radioactive liquid storage tanks were eliminated from further evaluation as shown in Appendix E and Appendix J, respectively, of the Final Operable Unit 9-04 Work Plan (Lee et al. 1996). Only five of the 90 facilities at ANL-W were retained for further qualitative evaluation in the Operable Unit 9-04 RI/BRA. The new information presented in Sections 5.7.2.1 through 5.7.2.5 present the procedures that ANL-W has in place to deal with a release if a release were to occur. The probability of a potential releases at these facilities are reduced by the ANL-W facility-specific procedures and controls that are currently in place. Changes in the status of these facilities will be evaluated when they occur to ensure that no releases to the environment occur, or if a release does occur, it is properly assessed and remediated, if necessary. Ultimately with an operating facility, the probability of having a release can never be completely eliminated. However, DOE believes that with strict adherence to the facility-specific operating procedures and controls, the probability of a release occurring is reduced to acceptable levels.

5.13 References

Arnett R. C., R. C. Martineau, M. J. Lehto, 1990, *Preliminary Numerical Model for Flow of Radionuclide Transport in the Snake River Plain Aquifer Near the Idaho National Engineering Laboratory,* EGG-WM-8820, Idaho National Engineering Laboratory, Idaho Falls Idaho

Amdur, M. O., T. Doull, and C. D. Klaassen, 1991, "Casarett and Doull's Toxicology," *The Basic Science of Poisons,* 4th ed., Pergaman Press.

Armitage, R., and R. Doll, 1961, *Stochastic Models for Caranogenesis,* In: Proceedings of the Fourth Berkeley Symposium on Mathematical Statistics and Probability.

ATSDR, 1990a, Agency for Toxic Substance Disease Registry, *Toxicological Profile for Plutonium.*

ATSDR, 1990b, Agency for Toxic Substance Disease Registry, *Toxicological Profile for Uranium.*

ATSDR, 1990c, Agency of Toxic Substances and Disease Registry, *Toxicological Profile for Acrylonitrile*, US Public Health Service, February 16.

ATSDR, 1990d, Agency for Toxic Substance Disease Registry, *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (Draft)*, February.

ATSDR, 1992a, Agency for Toxic Substance Disease Registry, *Toxicological Profile for Vanadium.*

ATSDR, 1992b, Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Barium.*

ATSDR, 1993a, Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Cadmium*, PB93-182418, April.

ATSDR, 1993b, Agency for Toxic Substance Disease Registry, *Draft: Toxicological Profile for Zinc*, December.

ATSDR, 1993c, Agency for Toxic Substances and Disease Registry, *Toxicological Profile for (Di-2-ethylhexyl-phthalate*, PB92-1824000, April.

Baca, R. G., S. O. Magnuson, H. D. Nguyen, and P. Martin, 1992, *A Modeling Study of Waterflow in the Vadose Zone Beneath the Radioactive Waste Management Complex, Idaho National Engineering Laboratory*, EGG-GEO-10068, Idaho National Engineering Laboratory, Idaho Falls Idaho.

Baes, C. F., III, et al., 1984, *A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides Through Agriculture*, U.S. Department of Energy, ORNL-5786, Oak Ridge National Laboratory, Oak Ridge, TN.

BEIR IV, 1988, *Health Effects of Radon and Other Internally Deposited Alpha-Emitters*, Committee on Biological Effects of Ionizing Radiations, National Research Council, National Academy Press, Washington, D.C.

Burns, D. E., 1995, *WAG-2 OU 2-13 Comprehensive Remedial Investigation/Feasibility Study Baseline Risk Assessment Technical Memorandum*, INEL-95/0342, November.

Cornfield, J., 1977, *Carcinogenic Risk Assessment*, Science, 198:693-699.

Crump, K. S., H. A. Guess, and K. L. Deal, 1977, *Confidence Intervals and Tests of Hypotheses Concerning Dose Response Relations Inferred from Animal Carcinogenicity Data, Biometrics*, 333:437–451.

DOE, 1994, *Incorporating Ecological Risk Assessment into Remedial Investigation/Feasibility Study Work Plans*, U.S. Department of Energy Memorandum, July.

DOE-ID, 1992, *Track 1 Sites: Guidance for Assessing Low Probability Hazard Sites at INEL*, DOE/ID-10340(92), Rev. 1, July.

DOE-ID, 1994b, *Track 2 Sites: Guidance for Assessing Low Probability Hazard Sites at the INEL,* DOE/ID-10389, Rev. 6, January.

DOE-ID, 1995, *Long-Term Land Use Future Scenarios for the Idaho National Engineering Laboratory,* DOE/ID-10440, August.

EG&G Idaho, 1991, *Preliminary Draft Remedial Investigation Volume 1; Perched Water System Remedial Investigation/Feasibility Study for the Test Reactor Area of the Idaho National Engineering Laboratory, Operable Unit 2-12,* EGG-WM-10002, EG&G Idaho, Idaho Falls Idaho.

EPA, 1985, *Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Office of Water Regulation and Standards,* June.

EPA, 1986a, *Guidelines for Carcinogen Risk Assessment,* Federal Register, 51:33992-34003.

EPA, 1989, *Soil Sampling Quality Assurance User's Guide (Second Edition),* EPA/600/8-89/046, March.

EPA, 1989b, *Risk Assessment Guidance for Superfund: Volume I, Human Health Evaluation Manual, Part A, Interim Final,* EPA/540/1-89/002, Office of Emergency and Remedial Response, Washington, DC, December.

EPA, 1991, *Supplemental Risk Assessment Guidance for Superfund,* EPA Region 10, August.

EPA, 1991b, *Risk Assessment Guidance for Superfund: Volume I-Human Health Evaluation Manual (Part B, Development of Risk-Based Preliminary Remediation Goals),* Office of Emergency and Remedial Response, Publication 9285.7.01B, U.S. Environmental Protection Agency, Washington, D.C.

EPA, 1991c, *ECO Update, Ecological Assessment of Superfund Sites: An Overview,* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Intermittent Bulletin 1(2):1-8, Washington, D.C.

EPA, 1992a, *Framework for Ecological Risk Assessment,* PB93-102192, EPA/630/R-92/001, U.S. Environmental Protection Agency, ORD/Risk Assessment Forum, February, 55 pp.

EPA, 1992b, *Dermal Exposure Assessment: Principles and Applications,* Office of Health and Environmental Assessment, Washington, D.C.

EPA, 1992c, *Supplemental Guidance to RAGS: Calculating the Concentration Term,* Office of Solid Waste and Emergency Response, Washington, D.C., EPA Publication 9285.7-081, May.

EPA, 1993, *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons,* Environmental Criteria and Assessment Office.

EPA, 1994a, Health Effects Assessment Summary Tables, EPA/540/R-94/020, November.

EPA, 1994, OSWER Directive 9355.4-01FS, *Guidance for Remedial Actions at Superfund Sites with PCB Contamination.*

EPA, 1994, *The Hydrologic Evaluation of Landfill Performance (HELP) Model; Engineering Documentation for Version 3,* EPA/600/R-94/168b.

EPA, 1995a, Health Effects Assessment Summary Tables, EPA 540-R-94-020, November.

EPA, 1995b, *Assing Dermal Exposure from Soil,* EPA/903-K-95-003, December.

EPA, 1995c, Drinking Water Regulations and Health Advisories, May 1995.

Gilbert, R. O., 1987, *Statistical Methods for Environmental Pollution Monitoring,* Van Nostrand Reinhold, New York, N.Y.

Golden Software Inc., 1996, SURFER for Windows Version 6, Golden Software Inc., Golden Colorado.

LITCO (Lockheed Idaho Technologies Company), 1995, *Idaho National Engineering Laboratory Management Guidance for Disposal of Wastewater,* INEL-95/307, November.

LITCO, 1996, *White Paper in the Food Crop Ingestion Exposure Route,* Rev. 2, INEL-95/0275, May.

Magnuson S. M., A. J. Sondrup, 1996, *WAG 7 Subsurface Pathway Flow Model Development and Calibration,* INEL-96/0297, Idaho National Engineering Laboratory, Idaho Falls Idaho, August.

Maheras, S. J., A. S. Rood, S. O. Magnuson, M. E. Sussman, R. N. Bhatt, 1994, *Radioactive Waste Management Complex Low-Level Waste Performance Assessment,* EGG-WM-8773, Idaho National Engineering Laboratory, Idaho Falls Idaho, May.

Mantel, N., N. R. Bohidar, D. C. Brown, J. L. Ciminera, and J. W. Tukey, 1971, *An Improved "Mantel-Brown" Procedure to Safety Testing of Carcinogens,* Cancer Res., 35:865–872.

Martin, K. L., C. J. Barnard, A. L. Freeman, M. R. Groh, K. T. Kissell, S. J. Lord, G. L. Olson, P. D. Randolph, R. N. Wilhelmsen, 1992, *Preliminary Assessment of Surface Soils at Active EG&G Idaho Facilities Data Document,* EGG-ESQ-9225, Rev. 1, May.

NRC (U.S. Nuclear Regulatory Commission), 1983, *Radiological Assessment: A Textbook on Environmental Dose Analysis,* J. E. Till and H. R. Meyer, editors, NUREG/CR-3332, ORNL-5968, September.

National Toxicology Program (NTP), 1985, *Twenty-six week subchronic study and modified mating trial in F344 rats. Butyl benzyl phthalate. Final Report,* 12307-02-03, Hazelton Laboratories America, National Toxicology Program, unpublished study, cited in IRIS.

Press, W. H., B. P. Flannery, S. A. Teukolsky, and W. T. Vetterling, 1992, *Numerical Recipes: The Art of Scientific Computing,* Cambridge University Press, Cambridge England.

Rood, A. S., 1994, *GWSCREEN: A Semi-Analytical Model for the Assessment of the Groundwater Pathway from Surface or Buried Contamination: Version 2.0, Revision 2.0,* Theory and User's Manual, EGG-GEO-10797, June.

Rood, S. M., G. A. Harris, and G. J. White, 1996, *Background Dose Equivalent Rates and Surficial Soil Metal and Radionuclide Concentrations for Idaho National Engineering Laboratory,* INEL-94/0250, Rev. 0, February.

Sax, N. I., and R. J. Lewis, 1989, *Dangerous Properties of Industrial Materials,* New York, van Nostrand Reinhold.

Singh, A. R., W. H. Lawrence, and J. Autian, 1972, "Teratogenicity of phthalate esters in rats," *Journal of Pharmaceutical Science*, 61: 51-55.

Sittig, M., 1981, *Handbook of Toxic and Hazardous Chemicals*, Noyes Publications, Park Ridge, NJ.

VanHorn, R. L., N. L. Hampton, and R. C. Morris, 1995, *Guidance Manual for Conducting Screening Level Ecological Risk Assessment at the INEL*, INEL-95/0190, Idaho National Engineering Laboratory, Lockheed Idaho Technologies Company, Idaho Falls, ID, April.